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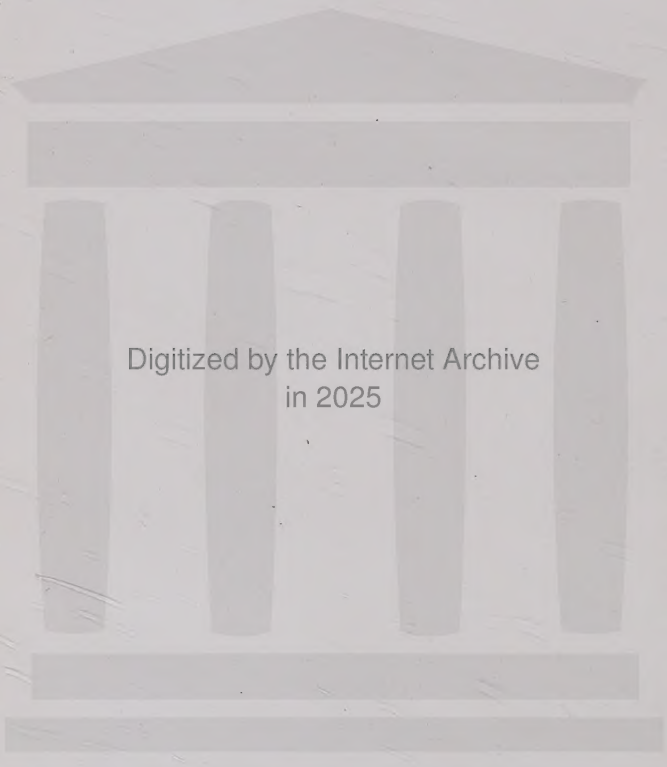

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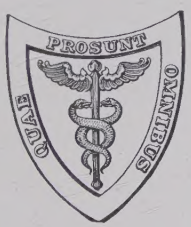

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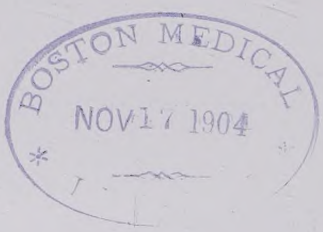

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PREFACE.

MEDICAL progress is so rapid in our day that Manuals have special value, in that they may be published in the shortest possible time, and thus place before the reader the most recent advances in Medicine. Moreover, a brief epitome presents the subject to the busy practitioner and student in a form more readily accessible than is possible in a lengthy treatise.

All the standard authors of the day have been consulted in the preparation of this book, and the author trusts that it will prove a valued assistant to the student.

GEORGE E. MALSBARY.

CINCINNATI, OHIO.

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PRACTICE OF MEDICINE.

CHAPTER I.

INFECTIONS.

DISEASES CAUSED BY VEGETABLE PARASITES.

SEPTICÆMIA (Pyæmia; Septico-pyæmia; Sepsis).

Definition: *Septicæmia* is a septic infection, due to the presence of the products, toxins, of pyogenic (pus-producing) micro-organisms in the blood and tissues of the body. *Pyæmia* is an infection of the blood and tissues of the body by pyogenic micro-organisms. In septicæmia the symptoms of blood-poisoning predominate; in pyæmia, the symptoms of metastatic abscesses. A combination of septicæmia and pyæmia constitutes *septico-pyæmia*. *Usually all these conditions are included under the term septicæmia* when there is general infection of the blood. *Localized infections* or inflammations have received special names—*e. g.*, meningitis, pleuritis, peritonitis, arthritis, metastatic abscess, etc. The condition caused by the absorption of toxins only, from a localized septic infection, is known as *septic toxæmia*.

Etiology: The micro-organisms most frequently encountered in *septicæmia* are the *staphylococcus pyogenes aureus*, which produces chiefly circumscribed abscesses, and the *streptococcus pyogenes*, which produces extensive suppuration. Puerperal septicæmia (*puerperal fever*) is usually caused by the streptococcus pyogenes. Other micro-organisms which act as etiological factors are the staphylococcus pyogenes albus, the staphylococcus pyogenes citreus, and the micrococcus pyo-

genes tenuis. More rare are the pneumococcus, the bacillus coli communis, and the bacillus of malignant œdema.

The micro-organisms gain entrance to the circulation through some break in the surface of the body, which may be caused by traumatism (wounds, parturition), or through a lesion produced by some other infection (tuberculosis, small-pox, dysentery, gonorrhœa), thus constituting a *secondary infection*. Cases of *cryptogenetic sepsis* are those in which the local depot of infection may not be discovered. Cases of discoverable lesion are *phanerogenetic*.

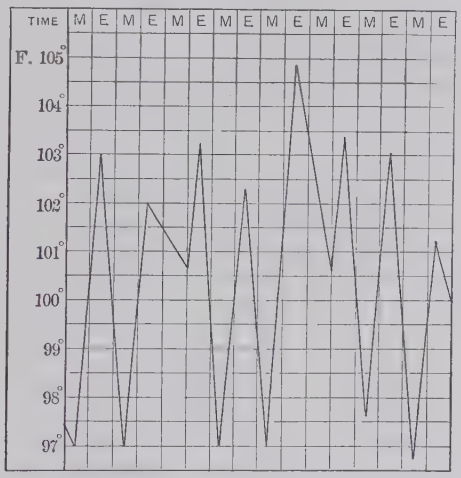
Infection may be conveyed by insects—*e. g.*, bedbugs, roaches, and flies (Coplin).

In order that septicæmia may be produced the resistance (immunity) of the body to the invasion of micro-organisms must be overcome, as in other infections. Special *obstacles to infection* are found in the skin, mucous membranes, the serosæ, in the small-cell infiltration, lymph-structures, lymph-vessels and -glands, the thymus gland, and bone-marrow; in the *excretory organs*, kidneys, liver, and intestine; and in the *blood*, where resistance is offered by both the corpuscles and the serum. No protective principle has been isolated from the blood. It has been proved experimentally that the immunity to infection depends largely upon the degree of alkalinity of the blood. Immunity is lessened by fatigue, starvation, exposure to cold, more especially to impure air, bad hygiene, and by toxæmia (Bright's disease) and anæmia.

Septicæmia—symptomatology There may be a preceding infection or traumatism. Infection by the pyogenic micro-organisms is announced by *chills and fever*, 103°–104° F.; and these are repeated with each new invasion. The elevation of temperature is accompanied by *nervous symptoms*, depression, headache, dizziness, and sometimes vomiting. There is profuse *sweating*. The fever recurs daily or every other day, in chronic cases often at longer intervals, with varying intensity, and is characterized by its irregularity, constituting the "*streptococcus-curve*" (Fig. 1), and by its resistance to treatment with quinine or the salicylates. The temperature may be above or below normal during the intervals. *Examination of the blood* sometimes reveals the micro-organism caus-

ing the disease, and usually shows an increase of white blood-corpuscles (leucocytes), a decrease of red blood-corpuscles (erythrocytes), and an increase of blood-plaques. Soon there is pain in the joints, which are swollen and tender. The spleen is enlarged. The skin, at first pale, becomes icteric. The

FIG. 1.



Streptococcus-curve from a case of phthisis.

pulse is rapid, often 120–140 per minute, weak and irregular. The typhoid state ensues. Evidences of metastatic affection may be found, especially in the organs having end-arteries—the skin, eyes, heart, kidneys, and brain.

Septicæmia—diagnosis: This rests upon the infection, chills and fever, the sweating, the frequency of the pulse out of proportion to the temperature, and the metastases.

The **differential diagnosis** concerns chiefly:

1. *Typhoid fever.* Both diseases may show an eruption of rose-colored spots, fever, diarrhœa, enlargement of the spleen, and bronchitis. Typhoid fever has a characteristic temperature-curve, very different from the see-saw, irregular "streptococcus-curve." The typhoid state is present much earlier in typhoid fever than in septicæmia. The presence of the

diazo-reaction (Ehrlich) in the urine, and the positive reaction of the blood to the blood-test for typhoid fever (Widal), would make the diagnosis of typhoid fever almost, if not quite, absolute. An examination of the blood may reveal the micro-organism causing septicæmia; but such micro-organism may be present as a secondary infection in cases of typhoid fever. The diseases may co-exist. Retinal hemorrhages, arthritic affections, and mitral lesions would speak for septicæmia.

2. *Malaria;* in which there is a distinct periodicity of fever, usually not to be found in septicæmia. A therapeutic test may be made with quinine, which has absolute control over malaria but no permanent influence over septicæmia. The presence of *plasmodium malariæ* in the blood speaks positively in favor of malaria.

3. *Miliary tuberculosis*, which may sometimes be differentiated by finding the tubercle bacillus in the secretions and excretions. Often there is tuberculosis of the lungs, lymph-glands, spine (caries), or hip-joint (hip-joint disease). Obscure cases may be cleared up by a test-injection of tuberculin. Septicæmia often exists in cases of tuberculosis as a mixed infection.

4. *Cerebro-spinal meningitis:* Opisthotonos, hyperæsthesia, constipation, and the occurrence of the disease in the colder months, in soldiers and children, would speak for cerebro-spinal meningitis rather than septicæmia.

5. *Endocarditis*, which presents evidence of heart-disease in enlargement and bruit. Ulcerative endocarditis is an expression of septicæmia.

6. *Uræmia*, which shows more severe headache, with twitchings, convulsions, and coma. In uræmia, there are œdema, albuminuria, and tube-casts; there is often also hardness of the arteries.

7. *Affections of the joints*, rheumatic, post-scarlatinal, or gonorrhœal. These furnish the evidence or history of rheumatism, scarlatina, or gonorrhœa, in the absence of the "streptococcus-curve" or metastatic abscesses. Affection of the joints may be an expression of septicæmia as a secondary infection.

Septicæmia—prognosis: The prognosis is grave in all cases; but even bad cases may recover. In general the prognosis depends upon the possibility of removing or destroying centres or depots of infection. Mixed infection; or infection with streptococci, usually gives a more grave outlook than infection with staphylococci or pneumococci. The gravity of the case may be measured approximately by the height of the fever, the weakness of the heart, and the nervous symptoms.

Prophylaxis: The prevention of septicæmia calls for asepsis (surgical cleanliness) and antisepsis in the treatment of wounds. Pus, whenever and wherever recognized, should be evacuated.

Septicæmia—treatment: Bichloride of mercury solution (1 : 1000 or 1 : 2000) or carbolic acid solution (1 : 40 or 1 : 60) may be used in the treatment of wounds. The actual cautery, thermo-cautery, or galvano-cautery is sometimes useful. Cryptogenic cases are sometimes cleared up by the deep urethral injection of a strong solution of protargol or nitrate of silver, gr. xx to ʒj; or by a curettement of the uterus or the removal of a diseased ovary; or by the relief of a mastoid disease by operation. The entrance of infection through the respiratory tract may be combated by the use of antiseptics—boric acid, lactic acid, subsulphate of iron, etc. Intestinal antisepsis may be secured by the use of calomel, salol, β-naphthol, or ichthalbin.

The frequent failure of *serum-therapy* in the treatment of septicæmia is probably due to the fact that streptococcic serum protects only against infection by the particular variety of streptococcus from the culture of which the serum has been immunized. This is only what should be expected, as a large number of organisms which differ widely in virulence and other characteristics are included under the term streptococci. Protection has been secured against two or three varieties by immunizing against the two or three germs. The streptococcic serums rapidly deteriorate in the vials and soon become worthless. Marmorek reports results obtained from the use of antistreptococcic serum that are in a general way encouraging.

Sometimes excellent results, and almost always temporary

improvement, may be obtained by venesection and infusion of normal saline solution.

Alcohol, best in the form of whiskey or brandy, may be given in large quantities. In septicæmia alcohol does not readily produce toxic effects. It is supposed to increase the number of leucocytes and to neutralize toxins. Quinine, gr. v, may be given every two to four hours. Toxins in the blood may be neutralized by the use of the salicylates, iodine, mercury, bromine, or arsenic.

Fever and pain may be relieved by phenacetin, or in the presence of great weakness by lactophenin. Fever that becomes dangerously high may be controlled by hydrotherapy, sponging with cold water. Affections of the heart, joints, meninges, pleura, or peritoneum call for application of the ice-bag. Obstinate pain and sleeplessness demand the use of opium.

ERYSIPELAS (Saint Anthony's Fire; Rose, Wundrose, Rothlauf (German); Erysipele (French)).

Definition: An acute infection caused by the *streptococcus erysipelatis*, characterized by inflammation of the skin and lymphatics, fever, gastric disturbance, and symptoms on the part of the nervous system.

History: Erysipelas was recognized by Hippocrates and the early medical writers, but they did not know the cause of the disease. Henle (1840) attributed the disease to minute vegetable organisms. Trousseau (1848) pointed out that there must be a lesion as a starting-point of the infection. The organism now held to be the cause of the disease was discovered in the skin by Koch and Fehleisen (1881) independently of each other. The *streptococcus erysipelatis* was isolated and cultivated by Fehleisen, who also made therapeutic inoculations in man, and thus definitely established this organism as the cause of erysipelas.

Etiology: The essential factor in the causation of erysipelas is an inoculation with the *streptococcus erysipelatis*, which can occur only through a lesion. The abrasion through which the streptococcus gains entrance to the body may be so mi-

nute as to have entirely disappeared by the end of the period of incubation. Such cases were formerly termed idiopathic. The streptococcus erysipelatis bears a close resemblance to the streptococcus pyogenes; and some believe them to be identical, or that the streptococcus erysipelatis is a variety of the streptococcus pyogenes. Erysipelas has been produced in man, as a therapeutic measure, in the treatment of sarcoma and carcinoma, by inoculation with streptococci obtained from peritoneal pus (Petruschky). The streptococcus erysipelatis may be carried by third persons or things (fomites). Crowding favors contagion, and succeeding attacks render the individual more susceptible to infection.

Erysipelas—symptomatology: The period of *incubation* is from two hours (Heiberg) to two weeks (Echlier); usually one or two days. This period may show no symptoms. There may be more or less malaise, anorexia, and lassitude, such as are common in the infectious diseases.

The first striking symptom, as a rule, is a *severe chill* lasting from a few minutes to one or two hours, usually about half an hour. With the chill there may be nausea, sometimes vomiting. There is *fever*, 102°–105° F.; the pulse is frequent. These symptoms continue a day or longer, with anorexia, uneasiness in the epigastrium, malaise, *headache*, *vertigo*, and possibly delirium. The urine is scanty; the skin dry and hot. Before the eruption appears there is often a feeling of tension and fulness, sometimes pricking or itching, in the affected part.

The *eruption* appears upon the *face*, as a rule, presumably because the skin of the face is tender and more exposed to infection. The eruption is rose-colored, frequently resembling an ordinary erythema. The skin is swollen and œdematous. The eruption, starting usually from the nose, ear, eyelids, or scalp, may extend to other parts, but *avoids the chin;* likewise, starting from the breast, the eruption *will not appear over the ensiform cartilage.* Variations in the course of the eruption have given rise to the terms: *erysipelas migrans*, ambulans, serpens, or wandering erysipelas, when the disease affects large areas and continues to advance at one part while it disappears at another; *erratic* or *multiple ery-*

sipelas, in which there are a number of lesions, caused by infection (inoculation) in several places at about the same time; *erysipelas fixum*, fixed erysipelas, a localized mild erysipelas, a term applied by some to an inflammation around chronic ulcers, which, however, is usually not a true erysipelas.

Œdema causes often marked deformity of the eyes, nose, ears, and scalp. The infiltration of the scalp may cause the hair to fall out; to be restored, as a rule, upon the disappearance of the disease.

The eruption may involve the *mucous membrane* of the ear, nose, fauces, pharynx, and larynx, less frequently of the vagina and uterus, either by extension from the skin or primarily.

During the height of the disease there are insomnia, more frequently somnolence, and coma, with more or less delirium. There is constipation, the tongue is coated, and the spleen enlarged. When the fever is high there may be albuminuria.

The general symptoms are probably due to *toxæmia*. The streptococcus erysipelatis has not been found in the blood, although it has been found in the urine (Cerné). Others claim to have found the coccus in the blood, in which case the general symptoms would be due to a "specific" septicæmia.

Complications: *Mixed infection* is not uncommon in erysipelas. *Septicæmia* (*ulcerative endocarditis*) is a frequent complication. Occasionally there is a complicating dermatitis, seborrhœa, an affection of the scalp; abscess of the skin and superficial lymphatic glands; gangrene; *albuminuria*, almost constant after fifty; nephritis, which rarely becomes chronic; pneumonia, bronchitis, pericarditis, meningitis, icterus, dysentery, enterorrhagia, ulcer of the duodenum, peritonitis, affections of the joints, parotitis, conjunctivitis, keratitis, amaurosis, panophthalmia, and paralysis.

Erysipelas—diagnosis: The peculiar *inflammation* with *œdema* and *deformity*, the course of the disease, the general symptoms, the presence of the streptococcus erysipelatis, and a lesion of the surface, with a return of the tissues to a normal condition upon subsidence of the inflammation, are

characteristic. Diagnosis prior to the eruption is difficult or impossible.

The *differential diagnosis* concerns chiefly the erythemata, drug-eruptions (antipyretics, copaiba), malignant pustule, and malignant œdema.

Prognosis: Erysipelas is self-limited, and as a rule disappears leaving no trace. Healthy adults rarely die (Osler). Individuals debilitated by age, disease, or the abuse of alcohol may succumb. In protracted cases death may be caused by exhaustion. Infection in the first months of life, usually occurring at the *umbilicus*, is very grave. Complications may increase the gravity of the prognosis.

Prophylaxis: The streptococcus should be destroyed. To this end dressings, bedding, and clothing that come in contact with cases of erysipelas should be burned or thoroughly sterilized. Contaminated instruments and utensils should be boiled from five to ten minutes. The room and furniture, and all articles that cannot be subjected to the action of fire or steam, may be sterilized by the use of formaldehyde, solutol, or bichloride of mercury, or by prolonged exposure to fresh air.

Erysipelas—treatment: Mild cases, in healthy adults, may require no treatment. The diet should be light and nutritious; best milk, then soups, gruel, etc. Constipation may be relieved by calomel and Carlsbad salt. If necessary, the patient should be supported with alcohol. Otherwise internal medication has little effect upon the disease further than the relief of symptoms. Chloral may be given for nausea and vomiting. If the eyes are affected, the patient should be kept in a darkened room.

In the way of local treatment, many cases do well with only wet compresses or inunctions. Active treatment seeks to destroy the streptococcus erysipelatis. Solutions of carbolic acid (3–5 per cent.) or of bichloride of mercury (1 : 1000) are in common use. Carbolic acid, bichloride of mercury, and the biniodide of mercury may be used hypodermatically in 2 per cent. solutions. The injections should be made just outside of the area of inflammation, where the streptococcus erysipelatis is to be found. Unfortunately, the injections may

not be used in the face, where the disease most frequently appears. Strapping the surface with adhesive plaster has been reported to give good results, as does also the application of collodion and ichthyol. The employment of serum-therapy must be considered *sub judice* (see Septicæmia).

CEREBRO-SPINAL MENINGITIS (Epidemic Meningitis; Leptomeningitis; Cerebro-spinal Fever; Spotted Fever; Malignant Purpuric Fever; Petechial Fever).

Definition: An acute infectious disease, characterized by headache, hyperæsthesia, opisthotonos, herpes, and petechiæ, and affections of the special senses.

History: Cerebro-spinal meningitis was first accurately described by Vieusseux (Geneva, 1805). The disease appeared in the United States in 1806. Epidemics have been few. The disease is now pandemic; but the cases being often apparently isolated, recognition of the disease is sometimes difficult.

Etiology: The micro-organisms found in the exudate of cerebro-spinal meningitis are the *diplococcus intracellularis meningitidis* (meningococcus) of Weichselbaum and the *micrococcus pneumoniæ crouposæ*. These organisms are also present in the blood in some cases. The diplococcus intracellularis meningitidis is believed to be closely related to the micrococcus pneumoniæ croupos e. These micro-organisms are sometimes associated with, or supplanted by, *secondary infection* by the streptococcus pyogenes, staphylococcus pyogenes aureus, bacillus coli communis, the bacillus proteus, and rarely by other organisms. The avenue of entrance of the infectious agent is probably through the upper respiratory tract. *Cold*, *crowding*, and *childhood* are predisposing factors.

Cerebro-spinal meningitis—symptomatology: The period of *incubation* lasts from eight to ten days (Latimer).

The symptoms come on *suddenly*, without prodromata, with chill, vomiting, headache, and prostration. *Opisthotonos* begins to show itself in stiffness of the back of the neck, with tenderness in the course of a few hours. There is *hyperæsthesia*, usually in the lower extremities, which may become

general. Various eruptions, including petechiæ, occur, but are not characteristic. *Herpes* may be observed as early as the third day, and continue until after recovery, as a rule appearing first about the face. The *temperature* shows an early rise, 102° to 104° F., and an irregular course. The pulse is rapid, and later, with the temperature and respiration, shows great irregularity. *Headache* is persistent, and may be associated with vertigo. Usually there is *constipation*. The abdomen is boat-shaped. The urine is scanty, the bladder paretic. Bad cases show enuresis. The *urine* presents the diazo-reaction in severe cases.

The *blood* presents the changes found in suppurative inflammations. Leucocytosis is well marked during the active stage of the disease, and the amount of hæmoglobin is usually diminished. Epistaxis is frequently observed.

Complications: *Pneumonia*, catarrhal and croupous; various *pareses and paralyses;* affections of the *eye*, photophobia, conjunctivitis, neuritis, atrophy of the optic nerve, *blindness*, keratitis with ulcer, iritis, irido-choroiditis, panophthalmitis, and *anæsthesia* are the chief complications. Affections of the *ear*, suppuration of the middle and internal ear, perforation of the membranes, *deafness* from inflammation of the labyrinth, are often present. Sometimes there is pleurisy, pericarditis, or parotitis.

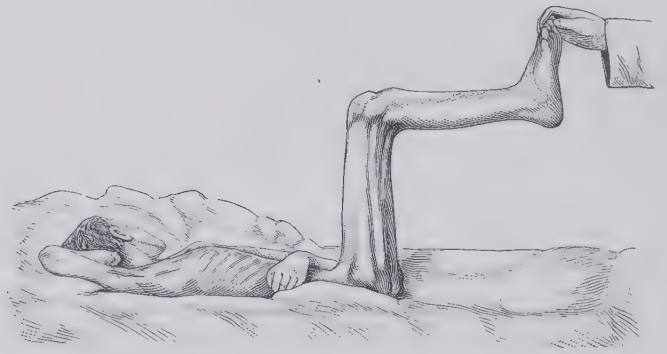
Cerebro-spinal meningitis—forms: (1) *Malignant* (foudroyant, siderant, fulminant, or apoplectic), in which death may take place in three and a half (Jewell) to thirty-six hours. (2) *Abortive*, including light cases, frequently unrecognized, in which convalescence begins after the symptoms have lasted from three to five days. (3) *Intermittent*, in which the intermissions are not so regular as in malaria. The temperature bears a closer resemblance to the streptococcus-curve in some cases of septicæmia, which may show more or less periodicity. The usual duration is from one to three weeks. (4) A *chronic* form has been described, which some believe to be the most frequent type (Heubner). Cases have been reported to last as long as fourteen weeks (Worthington). In this form there is a series of recurrences of fever.

Cerebro-spinal meningitis—morbid anatomy: Malignant

cases, if the patient die before exudation takes place, may present no characteristic changes. Exudation is most abundant on the cortex. The membranes may be thickened and adherent. The spleen shows more or less enlargement, according to the duration of the disease.

Diagnosis: The disease prefers *winter*, *soldiers*, and *children* (Pfeiffer). The diagnosis is usually easy when cerebro-spinal meningitis is epidemic. Cases of sudden death with symptoms of profound toxæmia should excite suspicion of cerebro-spinal meningitis in the malignant, foudroyant form. A *sudden onset*, with *chill*, and the presence of *headache*, *opisthotonos* (often only rigidity of the muscles of the neck), and *vomiting* are characteristic. Sometimes most information is to be obtained by *lumbar puncture* (Quincke), which may reveal the specific cause of the disease. The disease should not be mistaken for tubercular meningitis, malaria, tetanus, hydrophobia, smallpox, or typhoid fever.

FIG. 2.



Contracture of the knee-joint in the position of flexion, not admitting, without violence, extension beyond 135° with the thigh, while the patient is in the sitting posture, but which may be readily extended when the patient is in the erect or recumbent posture, is characteristic of meningitis (Kernig). (Fig. 2.)

Prognosis: The mortality varies with the epidemic—20 per cent. to 75 per cent. (Hirsch). Almost all malignant cases die. Abortive cases usually recover. The mortality in average cases is about 50 per cent. The outlook in childhood is graver than in adolescence. Most of the deaths occur in the first week of the disease, especially during the first three or four days. The prognosis is not so favorable in protracted cases, although recovery is possible.

Cerebro-spinal meningitis—treatment: The sick-room should be well ventilated; light, noise, and unnecessary visitors must be excluded. The diet should be light and nutritious. It may be necessary to use forced feeding. The bladder and bowels call for proper attention.

The chief remedy is opium, one grain every hour or two (Stillé), which may be given to relieve pain and spasm, and to protect the nervous system against the action of the poison of the disease. Large quantities of opium may be given without producing toxic effects. In cases of vomiting interfering with the administration of opium, morphine may be given subcutaneously, gr. $\frac{1}{3}$–$\frac{1}{2}$ for adults (v. Ziemssen). Later, *cold* (ice-bags) should be applied to the head and spine. Hot baths, 40° C., for ten minutes, give excellent results.

Vomiting and hiccough may be relieved by the internal use of hot water, cracked ice, milk and lime-water, soda, creosote, bismuth, chloral, or by morphine hypodermatically. Chloral may be given by enemata, if necessary, to enable the patient to retain food. A failing heart calls for stimulation. Alcohol, best in the form of whiskey or brandy, is well borne. Bloodletting and blisters have their advocates.

In extreme cases lumbar puncture or laminectomy and irrigation are justifiable.

RHEUMATISM.

The term "**rheumatism**" (ῥεῦμα, ῥέω, to flow) has come down to us from the humoral pathologists. The term "catarrh," which has the same derivation as rheumatism, with which it was synonymous, became confined to affections of the mucous membranes about the time of Ballonius (1600). "Rheumatism" became limited to diseases characterized by

pain about the bones, joints, and other structures than mucous membranes, which are not attributed to any special or specific cause. Later investigations have isolated gout, arthritis, trichinosis, syphilis, tuberculosis, and rickets. The term "rheumatism" is now used to cover at least five distinct affections:

Acute articular rheumatism,
Chronic articular rheumatism,
Gonorrhœal rheumatism,
Muscular rheumatism, and
Nodular rheumatism (see Arthritis Deformans).

Acute Articular Rheumatism.

Definition: An acute infectious disease, characterized by multiple arthritis.

Etiology: The disease is almost limited to the period of adolescence, fifteen to thirty-five years, and prefers fall and winter, when the weather is most changeable; but no season is exempt. The disease is rare before four or after forty years. Individuals most frequently affected are those exposed to changes of temperature—drivers, servants, bakers, sailors, and laborers. The disease is frequently ascribed to taking "cold." Acute articular rheumatism often occurs in the course of the infections, especially scarlet fever, dysentery, and septicæmia (puerperal). The disease is believed to be due to some infectious agent, probably closely related to the streptococcus pyogenes. Often the infectious agent seems to gain entrance to the body through the tonsils.

Symptomatology: The onset of the symptoms of acute articular rheumatism is often preceded by *angina*, especially *tonsilitis*, and *malaise*. Usually the disease begins suddenly with a *chill and fever*, reaching 102°–105° F. within a day. The pulse is usually above 100. There are more or less malaise and general distress. *Affection of the joints* is usually observed within the first twenty-four hours. The disease shows a preference for the *medium-sized joints*, especially the knee, ankle, and wrist; later the shoulder and elbow, and still later the fingers, and the vertebral and sterno-clavicular joints. Rarely there

may be involvement of the articulations of the maxilla, larynx, pelvis, and ribs. The joints become red and swollen. There may be subcutaneous œdema. The disease *flits from joint to joint*, often to return again to a joint previously affected.

There is *profuse sweating*, which lowers the temperature for a time. The perspiration is acid in reaction and sour-smelling. Often there are sudamina, especially in the absence of cleanliness.

Examination of the blood reveals marked *anæmia and leucocytosis*. The *urine* is usually reduced in quantity, concentrated, of high color, acid in reaction, and loaded with urates. The chlorides are diminished, and sometimes absent. The saliva may show an acid reaction and an excess of sulphocyanides.

Complications: The chief complication of acute articular rheumatism is usually on the part of the *heart:* pericarditis, endocarditis, or myocarditis. Some cases show hyperpyrexia, the temperature reaching 110°–118° F. Upon the part of the lungs there may be pneumonia or pleurisy. Some cases show delirium and coma; less frequently convulsions, rarely meningitis. Often there is chorea. The presence of sudamina has been mentioned. There may be a red miliary rash, scarlatiniform eruptions, purpura, often urticaria, and erythema. Rheumatic nodules are sometimes found upon the tendons and fasciæ.

Diagnosis: The affection of medium-sized joints, and especially the flitting from joint to joint, are characteristic points. Atypical cases and cases that do not respond readily to treatment should arouse the suspicion that they are not cases of rheumatism. Acute articular rheumatism must be separated especially from other forms of rheumatism, involvement of the joints in septicæmia, and gout and sarcoma.

Prognosis: Rheumatism has, in itself, a mortality of about 3 per cent. The remote effects are more dangerous. From one third to one-half of the cases have permanent heart-lesions. Sometimes the heart-lesions entirely disappear.

Acute articular rheumatism—treatment: The patient should wear flannel and sleep between blankets. The best article of diet is milk, which may be diluted with alkaline mineral

waters. Thirst may be relieved by free ingestion of fluid. Often relief may be obtained by fixing the joint—sometimes simply by wrapping the affected joint in cotton or hot cloths. Various liniments may be used, and are of value chiefly through massage and the application of heat.

Pain is sometimes relieved by the use of blisters or a light application of the Paquelin thermo-cautery. Salicin, salicylic acid, and the salicylates, for a time regarded as specifics, relieve pain and probably neutralize toxins. The oil of wintergreen, ℳxx in milk every two hours, often gives good results. The salicylates are probably best given with alkalies, potassium or sodium bicarbonate or iodide, in sufficient dosage to render and keep the urine alkaline in reaction. Severe pain may demand opium, best in the form of Dover's powder, or morphine. As a rule, antipyrin, or better phenacetin, salipyrin, or salophen will suffice.

Excessive fever (hyperpyrexia) may be controlled best by the cold bath. Tumultuous action of the heart may be relieved by application of the ice-bag.

Chronic Articular Rheumatism.

Occurrence and symptoms: Only exceptionally **chronic rheumatism** may result from acute rheumatism. As a rule, chronic rheumatism comes on insidiously, after the meridian of life, and *remains confined to the joint or joints first affected*. The disease is found especially among the poor—those most exposed to cold and damp. The affected joint is somewhat swollen, stiff, and painful. The pain is increased during damp weather or upon exposure to cold and damp. The joint may become ankylosed. Chronic rheumatism shows a preference for the *larger joints*—hip, shoulder, knee, wrist, and ankle.

Diagnosis: The age of the individual, the number of joints affected, longer duration despite medication, and the absence of sweating, high fever, or complications on the part of the heart, are important points in diagnosis, and serve to differentiate chronic from acute rheumatism.

Prognosis: Life usually is not shortened; but the outlook as to cure is not good. The disease is exceedingly obstinate to treatment.

Chronic rheumatism—treatment: Iodide of potassium is probably the best internal remedy. The salicylates may relieve the acute pain or exacerbations. Most may be accomplished by the local application of heat and friction. All sorts of liniments are recommended. Sometimes the use of blisters affords relief.

Often most may be accomplished by climato-therapy, especially by prolonged residence in a warm climate, or at least by wintering in such a climate, to avoid cold, damp weather; but few patients can afford such treatment.

Gonorrhœal Rheumatism.

Gonorrhœal rheumatism prefers the period of *adolescence*, the *male sex*, and the *knee-joint*. There may be involvement of the ankle and joints of the foot. Usually the affection of the joints is observed within three months after the gonorrhœal infection. The joints are greatly swollen. The specific cause is the gonococcus; or the pyogenic micro-organisms, as a secondary process (see Septicæmia).

The disease runs a **chronic course**, does not show sweating nor involvement of the heart, and when finally cured does not return nor leave deformity.

Treatment: Chronic gonorrhœa should receive attention, to prevent continuous infection. In the treatment of gonorrhœal rheumatism, most may be accomplished with heat, electricity, friction, and massage. Further treatment is the same as for chronic rheumatism.

Muscular Rheumatism—Myalgia.

Etiology: Many cases are caused by trauma, whereby muscular fibres are ruptured. Other cases are attributed to cold and exposure, which probably act by localizing some infection or poison. At least one infection, that by the trichina spiralis, is now described separately under *Trichinosis.*

Symptomatology: The only characteristic symptom is *pain*, which may vary in all degrees of severity and character, and is confined to the voluntary *muscles.* The pain is usually *relieved by pressure.*

The chief **varieties** of muscular rheumatism are: *occipito-frontal* rheumatism; *torticollis*, cervical rheumatism, stiff neck; *pleurodynia*, which is chiefly an intercostal rheumatism; and *lumbago*, one of the most frequent and painful forms. Affection of the muscles of the head is sometimes known as *cephalodynia*. The pain may be localized in the muscles about the shoulder and upper part of the back—*scapulodynia*, *omodynia*, and *dorsodynia*.

Diagnosis: Myalgia must be *differentiated* from the infections, especially smallpox, tuberculosis, syphilis, and septicæmia; and aneurism, caries of bone, and tumors must be excluded. The separation from neuralgia is sometimes difficult.

Prognosis: Usually good.

Treatment: The muscle should be put to rest—*e. g.*, by strapping the chest with adhesive plaster in cases of pleurodynia. Heat, friction, and electricity are probably the best remedies. Pain may demand phenacetin, antipyrin, or morphine. Lumbago is sometimes relieved by acupuncture. Some cases may be cut short by a hot bath early in the course of the disease. In chronic cases iodide of potassium is the best single remedy. In all cases a careful search should be made for the cause, which should be removed or properly treated.

INFLUENZA (Influenza (Italian, from *influence*); the Grip; La Grippe; Epidemic Catarrhal Fever; Chinese Catarrh (Russian); the Russian Disease (German and Italian); Italian Fever, Spanish Fever (French)).

Definition: An acute infectious disease, caused by the *influenza bacillus*, characterized by *catarrhal symptoms* on the part of the organs of *respiration* and *digestion*, and *nervous symptoms*, especially *prostration*.

Influenza—history: The disease was probably recognized by Hippocrates (Parks), and epidemics of this nature were recorded in the ninth century. In 1173 the disease seems to have been epidemic throughout Europe. The first accurate description is of the epidemic of 1510, when it is said scarcely a person escaped. The epidemic of 1557 spread westward from Asia to Europe and to America. The epidemic of 1647, which appeared first in Italy and France (1626–27),

is the first epidemic of the disease mentioned in American records (Noah Webster). Influenza is now pandemic.

Etiology: The specific infectious agent is the *influenza bacillus*. This organism has been found in all cases of influenza examined, often in pure cultures in the bronchial secretion, frequently in the pus-corpuscles. In fatal cases it has penetrated into the peribronchial tissue and even to the pleura, where pure cultures have been found in the purulent exudation. The influenza bacillus disappears, in cases of influenza, with the cessation of the purulent bronchial secretion. Positive inoculation-experiments have been secured in apes and rabbits (Pfeiffer). The bacillus has been found in the blood. (Canon). Kaufman found the influenza bacillus in a large number of telephone-receivers examined.

The *influenza bacillus* is aërobic, non-motile; grows upon glycerin-agar in the incubator, drop-like colonies developing in twenty-four hours, which are characteristic in that the *drops do not coalesce* (Kitasato). The bacillus may be stained best with dilute carbol-fuchsin, or Löffler's methylene-blue solution with heat. The ends of the bacilli are most deeply stained, which probably at first caused them to be mistaken for cocci. The influenza bacillus prefers a soil containing hæmoglobin, and requires a temperature that liquefies gelatin.

Influenza is highly contagious, and may be conveyed by fomites (clothing, third parties). Children seem somewhat less susceptible than adults. About one-fourth of cases occur in early life. Influenza has been reported in infants only a few days old, but is more frequent in the second half of the first year. The most susceptible period of childhood is from the eighth to the tenth year.

Influenza—symptomatology: The disease may show the usual *prodromata* of infection: malaise, languor, headache, etc. The period of *incubation* varies from a few hours to four days. Usually the *onset* is sudden, with symptoms on the part of the *respiratory* tract, the *gastro-intestinal* tract, and the *nervous* system.

The *respiratory* tract presents *catarrhal symptoms*, some fever, dryness, and swelling of the mucous membrane of the nose, increased secretion, and coryza. Often there is intense

bilateral bronchitis, and in children there is frequently *pneumonia*. Influenza-pneumonia is an unfortunate complication. Photophobia and lachrymation are frequently present.

On the part of the *gastro-intestinal* tract there are *nausea*, *dyspepsia*, vomiting, diarrhœa, and *icterus*, symptoms due to inflammation—catarrh—of the gastro-intestinal mucous membrane.

The *nervous* symptoms are supposed to be largely caused by toxins. The *spirits are depressed*, the patient experiences sinking sensations, and there is *prostration*. *Headache* is a constant symptom, usually frontal—*supraorbital neuralgia*. There are pains in the back and legs and general soreness. There may be drowsiness and somnolence or insomnia. Vertigo may be persistent and severe. Rarely there is cerebro-spinal meningitis as a complication. Not infrequently *tuberculosis* follows influenza, or is changed from a latent to an active process.

Influenza—diagnosis: The *respiratory*, *gastric*, and *nervous* symptoms are characteristic. Symptoms on the part of the respiratory tract, the gastro-intestinal tract, or the nervous system may predominate in a given case or epidemic. In doubtful cases an attempt should be made to disclose the bacillus of influenza, which may be readily cultivated upon glycerin-agar in the incubating-oven. At the end of twenty-four hours small transparent drop-like colonies may be recognized, which are characteristic in that they do not coalesce.

Prognosis: Death seldom occurs, except among the feeble, the aged, invalids, and young infants. The chief danger lies in the predisposition to other diseases, especially tuberculosis.

Prophylaxis: If it were generally known by the laity that "colds" are contagious, there would probably be fewer cases of influenza. Isolation of influenza-cases, to be of value, must be more complete than is usually practicable. The debilitated should not be exposed to infection.

Influenza—treatment: The strength of the patient should be supported and individual symptoms met. The disease is self-limited; but one attack does not secure immunity for any considerable length of time.

Early in the course of the disease, especially when gastro-

intestinal symptoms predominate, calomel or the saline purgatives may be used. A light "fever-diet"—milk, the gruels, beef-tea—should be observed. Individuals previously weakened by disease, age, or the abuse of alcohol may require the use of alcohol, whiskey, or brandy. In relief of symptoms, appeal may be made to the salicylates, salicylate of sodium, salol, best salipyrin, or the salicylate of cinchonidin, which causes less depression; lactophenin, phenacetin, antipyrin; morphine or opium, best in the form of Dover's powder.

The oil of eucalyptus has been used, especially by the English. Fürst, in the treatment of children, claims good results from the local use of the vapor of turpentine and menthol. Benzonapthol has been highly recommended by Huchard in the gastric form of influenza, 5 mgr. (gr. $\frac{1}{12}$) in pills, several times a day. Some advocate the use of quinine, gr. xv–xx, and others condemn its use.

Excessive fever may call for hydrotherapy, the sponge-bath; as a rule, however, hydrotherapy should not be used.

WHOOPING-COUGH (**Pertussis**; **Tussis Convulsiva**; **Keuchhusten, Kindhusten** (**German**); **Coqueluche** (**French**); **Tosse Asenine** (**Italian**)).

Definition: An acute infection, especially of childhood, characterized by paroxysms of convulsive cough, with usually a peculiar inspiratory "whoop," an inflammation of the nasal, laryngeal, and bronchial mucous membranes.

History: At first not distinguished from bronchitis, influenza, and croup. Recognized by the Greeks (Mason Good). Definitely described by Baillou (Paris, 1578). First monograph published by Danz (1791).

Etiology: The catarrhal stage of whooping-cough is supposed to be due to the action of micro-organisms. The paroxysmal stage (whooping) is probably caused by the poison (toxin) generated by the micro-organism. There is considerable evidence in favor of the bacillus discovered in the sputum by Koplik, of New York, as the specific infectious agent. The bacillus is about the size of the influenza bacillus.

Cohn and Neumann found in the sputum, at the end of a spasm, after washing with distilled water and staining with carbol-methylene-blue, diplococci and small chains of cocci. Ritter found the diplococcus tussis convulsiva in all of one hundred and forty-seven cases in which the sputum was examined. This diplococcus resembles the gonococcus, but differs from that organism in that it grows upon agar (Schlossman). Neumann could find the organism described by Ritter in only one out of eighteen cases examined. A similar organism has been described by Heubner as the intracellular meningococcus. Some observers hold that the cause is an amœba belonging to the protozoa. Kurloff found, in fresh, unstained sputum, amœbæ with fine granular protoplasm, provided with cilia and showing active movement. Kurloff believes these to be the infectious agent of the disease, and that the bacteria, which he also observed, are probably concerned in the secondary affections and complications of whooping-cough.

Contagion is usually by contact; but may be through fomites, especially handkerchiefs. One attack usually confers *immunity*. The disease shows a preference for children, especially the weakly, from six months to six years old.

Whooping-cough—symptomatology: The period of incubation varies from two days to two weeks. The symptoms of an acute *catarrh* of the air-passages then develop, and may last a few days or throughout the course of the disease. The paroxysm of cough is preceded by a distinct *aura*, which the patient soon learns to interpret as a forerunner of a spell of coughing. The cough is usually rewarded by the discharge of a small quantity of *mucus*. *Vomiting* is common. Soon there comes the characteristic "*whoop*," an audible inspiration following a spasmodic cough. The "whoop" is heard at the close of a series of coughs.

Gilbert recommends recording on a chart the coughing-spells, in suspicious cases. The duration of the paroxyms is noted, and also the length of time between paroxysms. There is a coughing-spell about once every hour in the daytime, and every half hour at night. The paroxysms consist of six or eight coughs, "beginning with a big, loud cough, and tapering

down to a mere 'hack.'" Gilbert represents the whooping-cough diagramatically thus:

C C C C c c——C C C C c c——C C C c c——etc.

The cough of simple bronchitis may be represented thus:

c - c - c - c - cc - c - c - c - c - c - ccc - c - c - c - c - cc.

In this way Gilbert claims to be able to make a diagnosis in the first week of whooping-cough, before the characteristic whoop is heard.

An *ulcer* may frequently be found upon the frenum of the tongue, due to friction against the lower incisors. Sometimes the ulcer is found in the absence of whooping-cough, and it may be absent in cases of whooping-cough.

The *paroxysmal stage* continues usually two to six weeks. The severity of the symptoms begins to diminish, as indicated by fewer paroxysms, and after ten days to several months health is restored.

Complications: The most frequent complication of whooping-cough is *broncho-pneumonia*. Less frequent is emphysema. Petechia, especially upon the forehead, ecchymosis of the conjunctivæ, epistaxis, and hæmoptysis may occur. Albuminuria may be found, but serious kidney-lesions are not common.

Diagnosis: The *history* of exposure is often of value. The *cough* not only persists, but increases despite treatment. The "*whoop*" is characteristic. Gilbert claims to be able to make the diagnosis by the character of the cough (see Symptomatology), even in the absence of the "whoop." There is evidence of *inflammation* of the nasal, laryngeal, and bronchial mucous membranes. An *ulcer* on the frenum of the tongue, the result of friction against the lower teeth, may usually be found, but is not pathognomonic. In doubtful cases measles may be excluded if there be no eruption by the fifth day. Whooping-cough often occurs during convalescence from measles.

Prognosis: The prognosis is usually favorable; not so good in the debilitated or in the negro race. Frequently tuberculosis has been observed to follow whooping-cough.

Prophylaxis: The patient should be isolated. Isolation is

difficult to secure in mild cases. At any rate, invalids and delicate children must not be exposed to contagion. The sputum should be destroyed.

Whooping-cough—treatment: Mild cases may call for no treatment. Often it is only necessary to treat the associated catarrh. For the paroxysms a number of remedies have been recommended. Should the paroxysms not exceed half a dozen per day special treatment may not be necessary. Where the paroxysms are troublesome antipyrin, gr. ij–iij for a child two years of age, often acts very well. Acetanilid, phenacetin, and lactophenin may be used in individual cases. Bromoform, gtt. ij–iv three or four times a day for a child three to six years of age, on sugar or in alcohol, has many advocates. Belladonna, from two minums of the tincture or gr. $\frac{1}{12}$ of the extract up to tolerance, given three or four times a day at two years of age, has stood the test of time. Quinine, gr. j or more, every two or four hours, for a child two years old, is largely used. Opium (paregoric) relieves the cough, secures sleep, and protects the nervous system. Chloral may be given to relieve vomiting and secure sleep. A change of climate sometimes becomes necessary.

Raubitschek attempted to determine whether or not whooping-cough is due to bacteria, by the local application of bichloride of mercury, 1 : 1000, to the tonsils, uvula, epiglottis, and adjacent mucous membrane. The application was made every day in severe cases, and every other day in mild cases. As a rule, improvement was noticed on the second or third day. In the paroxysmal stage the disease disappeared after four or five treatments.

Naegely advises grasping the hyoid bone, over the two greater cornua, and the larynx, and holding them from sixty to ninety seconds, as a means of cutting short the paroxysms. He believes the action is due to the induction of an inhibitory reflex.

Rothschild found the faithful use of tussol from the beginning of the disease caused the whooping-cough to be milder and shorter in duration. Some cases recovered in two weeks.

Koroleff found the disease entirely disappeared in three days in four cases treated with naphthalin vapor; while in five

other cases, treated in the same way, the course of the disease was unaffected.

Neumann used benzine vapor with good results; but found little value from the use of chloroform by inhalation. Rehfeld, on the other hand, used chloroform anæsthesia in a case of whooping-cough while setting a broken thigh-bone, and there was an immediate disappearance of the whooping-cough.

Mohn found the inhalation of sulphur fumes was followed by a reduction of the duration of the disease to eight to fourteen days. The sleeping-room was charged with sulphur fumes. Two or three treatments were sufficient.

Topical applications secure better results than inhalations. Bichloride of mercury solution, 1 : 1000, never fails to arrest the disease (Raubitschek, Gentile, Fede).

Oliphant secured good results from the local application of formalin.

Ditel uses the bromides during the paroxysmal stage, followed in a few days by the use of codein. For the fever Ditel uses antipyrin, and for the bronchitis terpin hydrate.

Binz recommended the use of quinine. The remedy may be used per rectum. Unruh advises the insufflation of quinine into the nose and pharynx.

Celli found vaccination sometimes followed by a cessation of whooping-cough. Bolognini believes vaccination justifiable as a therapeutic measure in children that have not been vaccinated.

In cases complicated by broncho-pneumonia, when many of the remedies usually employed in whooping-cough are contraindicated, the use of camphor has been advised.

Good results may be secured in bad cases by the use of resorcin, 2 to 3 per cent. solution, applied locally.

The patient should be in the open air as much as possible. Thorough ventilation of the apartments should be secured, even in cold weather.

MUMPS (**Epidemic Parotiditis**; **Epidemic Parotitis**; **Mompen** (**Danish**); **Schafskopf, Ziegenpeter** (**German**); **Oreillons** (**French**)).

Definition: An acute, infectious, contagious, epidemic disease, characterized by inflammation of the parotid gland, often

complicated by involvement of the testicle in the male; and of the breast, ovaries, and external genitals in the female.

History: Mumps was recognized in the earliest times. The disease was described by Hippocrates.

Etiology: Many observers have cultivated micro-organisms found in cases of mumps. Michaelis found diplococci resembling the gonococcus and meningococcus, but smaller. Inoculation-experiments have not succeeded in producing the disease.

Infection probably occurs through the duct of Steno. Most cases are preceded by, or associated with, *inflammation* of the mucous membrane of the *mouth* or *throat*. The epidemic nature of mumps is well known. Contagion usually requires *close contact*, although infection may be carried by third parties (fomites). Mumps show preference for the period of *childhood* and *early adolescence*, especially from the fifth to the fifteenth years. The disease seldom appears under two years; age is almost exempt. It has been suggested (Soltmann) that the *exemption* of infancy and age may be attributed to the duct of Steno being small in infancy and atrophied in age. The exemption of age may be largely due to the exhaustion of susceptible material, few reaching advanced age without protection by previous attack.

Males are attacked more frequently than females. Mumps prevails especially during the *cold* months. The disease may affect animals (dogs).

Mumps—symptomatology: *Incubation* may be as short as three days (Leitzen), or as long as six weeks (Nicholson); usually about two weeks. This period presents no symptoms, at least no characteristic symptoms, of the disease. Prodromal symptoms of infection,—malaise, headache, neuralgic pains, anorexia, slight fever, less frequently diarrhœa, vomiting, convulsions,—are present in about one-third of cases (Rilliet and Barthez). These symptoms last from a few hours to a few days, usually two to eight days.

With the onset there is usually a *chill* or chilly sensations; then *fever*, as a rule 101° F. or less, reaching during the course of the disease 102° F., exceptionally as high as 104° F.

Evidence of affection of the parotid gland is one of the

earliest and most characteristic symptoms. Usually there is *pain* in one of the parotids. The gland soon begins to *swell;* the swelling becomes extensive, causes the *ear* to be *displaced* upward, outward, and forward, and may cause the head to lean to one side. As a rule, the infection extends to involve both parotids. Often there is involvement of the testicle—*orchitis*—in the male. In females affection of the breast—*mastitis*—is common, also of the external genitalia, rarely of the ovaries, oöphoritis. Sometimes the attack is announced by otalgia, especially in children (Comby).

Complications are rare. Affection of the labyrinth may cause *deafness.* Other complications, especially on the part of the *brain,* may be caused by interference with the *circulation* or by *toxæmia.*

Mumps—diagnosis: The presence of an *epidemic* is an aid in diagnosis, which is usually easy. The onset of the disease with *enlargement of the parotid,* indicated by *swelling at the angle of the jaw* and with *displacement of the ear,* sometimes of the head, with *pain,* tenderness, and more or less *fever,* characterizes the disease. In some cases, such as those marked only by orchitis without the development of other symptoms of mumps, an absolute diagnosis may be difficult or impossible.

Prognosis: As a rule, good. Uncomplicated cases do not die. According to Laveran, the chief danger in the adult male is orchitis, which occurs in about two-thirds of the cases, and results in atrophy seven times out of ten. Thus impotence may follow double orchitis, which, however, is rare. Other unfortunate complications are mastitis, nephritis, otitis, and permanent deafness.

Mumps—prophylaxis: This calls for *isolation,* three weeks to a month, and *disinfection* (steam, formaldehyd) of the sick-room and of all articles which come in contact with the patient. Prophylaxis is exceedingly difficult, since the disease is often so mild in character, and since it may be disseminated during the period of incubation and for some time after the disappearance of symptoms.

Mumps—treatment: The treatment is symptomatic. The patient should be kept in the house, in bed, if the fever

is high. Sometimes aconite is given for fever. The *diet* should be fluid or such as may be swallowed without distress. The *tension* caused by the enlargement of the parotid may be relieved by hot or cold applications, as the patient may prefer, usually best by hot poultices, lard, vaseline, olive oil, or cocoa-butter. Gargles, as with hot salt water, are of very great value. If orchitis develop, the testicle must be supported, and later treated with the faradic current. The bowels should be kept open, best with a saline laxative or calomel. Often the patient may be made more comfortable by the use of Dover's powder or phenacetin.

Complications must be met by special treatment.

MEASLES: **Morbilli (Italian); Rubeola (Sauvages); Rougeole, Ruber (French); Masern (German); Masura (Sanscrit)).**

Definition: A very contagious acute infection, characterized by early catarrhal symptoms, coryza, and bronchitis, and later by a peculiar eruption.

Etiology: Doehle (1891) described bodies resembling *protozoa* in eight cases of measles. The observation lacks confirmation; but many believe that measles, scarlet fever, and smallpox may be due to organisms of this character. Canon and Pielicke (Berlin, 1892) discovered a short, thin *bacillus* in the blood of measles patients in fifty-six cases. The bacillus varies from 0.5 μ to the diameter of a red blood-corpuscle in length, and in culture is found in long threads. The discovery has been confirmed by some observers (Czajkowski, Grigorieff), while others (Barbier, Warschovsky) have failed to find the bacillus in cases of measles. Some observers believe the cause is a micro-organism that is too small to be recognized by the strongest known power of the microscope.

Measles shows a *preference for winter and spring*. The disease is very contagious, which explains the apparent liability of childhood, especially from one to five years. Comparative exemption of the first six months of life is probably due to freedom from exposure to infection. Individuals in later life are protected largely by previous attack. The children of mothers with measles show marked exemption from the disease.

Measles may be communicated through the nasal secretion, which explains the general belief that measles is contagious through the breath. The disease may be conveyed by third parties, clothing, etc. Measles is contagious throughout its course; probably during incubation, certainly during the prodromal stage.

Measles—symptoms: The period of *incubation* lasts from seven to eighteen days, during which there are no symptoms characteristic of the disease. Inoculation-experiments have placed the incubation at *ten days*. The eruption appears about two weeks after exposure.

Invasion: The patient has a *shivering fit*, possibly a chill. At this time there may be no noticeable fever, but soon the temperature rises to 100°–104° F., with symptoms on the part of the *stomach* and *nervous system*. There is *inflammation of the mucous membrane* of the eyes, nose, pharynx, and larynx, with severe *coryza*, *cough*, and *photophobia*. The mucous membrane of the cheeks is swollen to show the imprint of the teeth. There is *bronchitis*.

Usually during the second day the eruption appears, first as an *enanthem* upon the mucous membrane of the mouth, from one to five days before the exanthem appears on the skin. The former reaches its height just as the eruption on the skin is appearing, and then fades. Koplik describes the enanthem as minute bluish-white specks on a reddish punctuate area in beginning measles, and on a more diffused background in advanced cases. Microscopic examination of the spots reveals diplococci and epithelial cells. This enanthem is believed to be pathognomonic of measles. The breath has the odor of sour paste. The patient suffers general malaise and thirst. In some cases, especially in certain epidemics, dulness and somnolence appear among the prodromata.

From the third to the fifth day, usually about the fourth day, the *exanthem* appears, as a rule, first on the forehead, at the edge of the scalp, or behind the ears; later, around the eyes and mouth, and on the chin and neck. The eruption may be at first red and punctiform, or only a diffuse redness; but in a few hours small rounded red spots appear, separated by ap-

parently healthy skin. At first the spots disappear on pressure, to reappear when the pressure is removed. Later the spots no longer disappear on pressure.

The *eruption* gradually spreads from the forehead and sides of the face downward over the trunk and upper extremities by the seventh day, and over the lower extremities by the eighth day. Within about twenty-four hours after the first appearance of the exanthem, the eruption begins to disappear. Thus the eruption may vanish from the face before it appears on the lower limbs. With the disappearance of the eruption there is an improvement in the general symptoms, usually with a return to health in ten to fourteen days. After the eruption there is a *desquamation*, usually fine and branny. Desquamation is sometimes absent, especially in light cases.

In the absence of complications, an average case of measles presents approximately ten to fourteen days' incubation, three days' invasion, three days' progress, and three days' decline.

Measles—forms: In severe cases, *rubeola siderans*, the individual may be overwhelmed with the poison of the disease and die during the stage of invasion. On the other hand, in very light cases the patient may show little evidence of illness. Almost any of the symptoms may be present or absent in a given case. Thus there may be none of the symptoms of catarrh, *rubeola sine catarrho;* the eruption may be absent, *rubeola sine eruptione*, although probably some eruption is present in every case; or there may be little or no fever, *rubeola afebrilis*.

Hemorrhage may take place under the skin or from the mucous membrane of the urethra, vagina, nose, intestine, and other mucous membranes, or into the muscles and serous membranes, *rubeola nigra*, black measles. Such cases rarely occur in private practice, but may be found under bad hygienic surroundings.

The more important **complications and sequelæ** of measles are: bronchitis, broncho-pneumonia, croupous pneumonia, *catarrhal pneumonia*, *tuberculosis*, pleurisy, stomatitis, *noma*, *laryngeal stenosis*, diphtheria, enterocolitis, endocarditis, pericarditis, *headache*, *convulsions* (especially in children), delirium,

tubercular meningitis, paralyses, chronic conjunctivitis, iritis, blepharitis, keratitis, catarrhal or purulent otitis, and nephritis.

Measles—diagnosis: Diagnosis is usually impossible during the period of invasion, and is often very difficult during the prodromal stage of the disease. The absence of previous attack and the presence of other cases of the disease may aid in some cases.

In measles there is a *long prodromal stage*, with *fever* and *catarrh*, and later a peculiar *eruption*. The spots described by Koplik in the enanthem (see Symptomatology), are of especial value in the differentiation of measles from scarlet fever, simple aphthæ, rubella, and influenza. One of the most characteristic signs is the *early photophobia*, which is often of value in the differentiation from influenza.

Measles should be *differentiated*, especially from simple catarrh or coryza, hay-fever, scarlet fever, rubella, variola, typhus, roseola, papular erythema, and drug-eruptions (copaiba, quinine, antipyretics).

Prognosis: The prognosis of measles would be excellent if it were not for the complications and sequelæ of the disease. Should the temperature continue high after the appearance of the eruption on the fourth or fifth day, complications may be expected. The most dangerous of these is *tuberculosis*, which is often changed from latent to active. *Bronchitis* and *pneumonia* are responsible for many deaths.

Prophylaxis: One of the chief difficulties in prophylaxis is the fact that measles is contagious before the appearance of characteristic symptoms. The child should be isolated upon the first suspicion of the disease. Widowitz believes that epidemics of measles could be prevented by closing the schoolroom in which the first case occurs, from the ninth to the fourteenth day after the first appearance of symptoms, during which time all children from the room should be isolated from other children. The children could then return to school upon presenting the certificate of a physician. The disease may be carried by third parties and things (fomites). Quarantine should be continued two or three weeks after the onset of symptoms. The patient should then receive a bath and put on clean clothing. The room, and all articles which

have come in contact with the patient, should be sterilized. As a rule, exposure to fresh air is sufficient to destroy the contagious principle of measles.

Measles—treatment: The subcutaneous injection of serum obtained from convalescents from measles has been practised by a number of observers (H. Thompson, Weisbecker, Hubert, Blumenthal), with results more or less encouraging. Weisbecker injected serum, obtained from a convalescent, into a child nine months old, showing the prodromal stage of measles and whose brothers and sisters were taken with the disease. Ten grammes of serum were injected, with the result that the eruption was confined to certain parts. In four cases of pneumonia following measles two cases received the same dose, ten grammes, and the other two twelve and eighteen grammes. In each of these cases resolution occurred in a few days. In two of the cases there was a rapid disappearance of the fever; in six hours in one case; in twenty-four hours in the other.

The use of the serum of convalescents is believed to confer immunity, to cause the disease to run a milder course, to shorten the duration of illness, and even to cause a rapid disappearance of general symptoms.

Distressing symptoms or complications should be met, and the patient placed under good hygienic surroundings and supported until the disease has run its course. Mild cases may call only for the relief of thirst, cough, and photophobia. Thirst may be relieved by water, simple or acidulated, lemonade, or raspberry vinegar. Milk is the best food, diluted with water, soda-water, mineral water. Cough may be controlled by codein or small doses of Dover's powder. Laryngitis, stomatitis, and pharyngitis may be treated with antiseptic solutions. Severe laryngitis may call for the application of hot fomentations to the front of the neck. In membranous laryngitis, intubation, or tracheotomy is sometimes necessary. Bronchitis may call for expectorants, best apomorphin. Photophobia is relieved by shading the eyes with smoked glasses or screens, and the irritation may be reduced by the local application of solutions of morphine or atropine. The edges of the lids should be anointed with vaseline to prevent them

adhering during sleep. Earache is relieved by the evaporation of chloroform near the meatus, or by the instillation of hot water or a grain-to-the-ounce solution of atropine. Slight fever may be disregarded; fever above 103° F. may be met with the warm bath or, though more unpleasant, the cold bath (Fodor, Dieulafoy). The desired result may often be obtained by the use of antipyrin, phenacetin, and similar remedies in small doses, with beneficial effect upon the nervous symptoms so often present. Nervous symptoms may call for the bromides; sleeplessness, for trional, chloral. Diarrhœa may be controlled by bismuth and opium. Constipation should be carefully treated to avoid diarrhœa, best by enemata, sometimes by small doses of castor-oil or calomel.

RUBELLA (**Rotheln**; **German Measles**; **French Measles**).

Definition: A contagious acute infection of short duration, presenting mild catarrhal symptoms and a characteristic eruption.

Etiology: Rubella occurs especially at from *five to fifteen* years of age, although adults are often attacked. Previous attacks of measles or scarlet fever do not protect against rubella. Micro-organisms have been found in the blood (Klamann, Edwards), but have not been proven to be the cause of the disease.

Rubella—symptoms: *Incubation* lasts from five days to three weeks, probably the most variable of any of the acute infectious diseases. The *prodromal* stage is short, one-half to one day, often scarcely perceptible. During this period there may be some symptoms of inflammation of the mucous membrane of the respiratory tract, some malaise, headache, vomiting, diarrhœa, or constipation; but as a rule these symptoms are not marked. Often the period of eruption sets in without previous symptoms.

Forchheimer attaches importance to the *enanthem* of rubella, which he believes to be present in all cases as a macular, distinctly rose-red eruption upon the velum of the palate and uvula, extending to, but not onto, the hard palate. The spots are arranged irregularly, not crescentically, are the size of

large pinheads, at the largest, and are very little elevated above the level of the mucous membrane. During the process of involution, especially in mouths having a pale mucous membrane, there are sometimes left pigmented deposits, usually of a yellowish-brown color, in spots or streaks.

The *eruption* extends from the face to the feet in a day. The eruption of rubella may resemble the eruption of measles or of scarlatina—*rubella morbillosa*, *rubella scarlatinosa*. The eruption may fade from one part before attacking another part, or it may cover the entire body at one time. The color is usually a pale red. The *maculæ* are more or less elevated, smaller, and not arranged in groups as in measles. The red points seen in scarlatina are absent in rubella. The eruption lasts at the longest only three or four days. There is little or no fever. *Desquamation* may be absent, and when present is slight and resembles that of measles. *Complications* are rare.

Diagnosis: Rubella is distinguished by its *mildness*, the absence or slightness of prodromata and fever, the *enanthem*, the diffuse rose-red *rash*, and the enlargement of the cervical lymphatics early in the course of the disease.

Rubella should be differentiated especially from measles, scarlatina, syphilis, and drug-eruption.

Prognosis: Excellent.

Prophylaxis calls for isolation.

Treatment: Little or no treatment is required. Usually it is difficult even to keep the child in bed. Any unpleasant symptoms should be treated symptomatically.

SCARLET FEVER (**Scarlatina**; **Scharlach** (**German**); **Scarlatine** (**French**); **Scarlatto** (**Italian**)).

Definition: An acute, highly infectious disease, exhibiting a peculiar rash, angina, and fever.

Etiology: Scarlet fever is generally believed to be due to some micro-organism, probably a coccus—micrococcus, streptococcus, diplococcus—but as yet this has not been proven. Many observers would attribute the failure to find the specific infectious agent to the limited magnifying power of the microscope. The pus-formers are commonly present in the local

inflammatory processes, in the exudate in the throat, and in secondary affections and suppurations. A *denuded surface*, sore throat, wound, the puerperium, predispose to infection. Scarlet fever may be conveyed directly or by third parties, clothing, milk, mail (fomites). The vast majority of cases occur under the age of ten years. Liability to attack is greatest at five years; the greatest mortality is at three (Gresswell). The disease has been observed as early as the second day of life (Cortes). The geographical distribution and the individual susceptibility are less in scarlet fever than in measles and smallpox. One attack confers immunity.

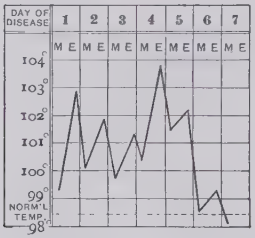
Fleming believes scarlet fever to be a local disease of the throat, and that the nephritis and dermatitis result from the attempt at excretion of the toxin.

Behle, of Frankfort, in a district in which "pigs' scarlatina" (English)—Rothlauf (German), Rouget (French)—had been previously unknown, found a severe epidemic of scarlet fever among children followed or accompanied by a disease among the pigs, marked by the symptoms of scarlet fever, including erythema, angina, albuminuria, and uræmia. Death in these cases was usually due to uræmia or angina. Characteristic lesions were found post-mortem in the kidneys. A previously healthy pig inoculated with the blood of a child suffering from severe scarlet fever, died a week later, and presented symptoms and post-mortem appearances identical with those of scarlet fever in man and the disease present in the other animals. The animals had probably been infected from children or from one another.

Scarlet fever—symptomatology: *Incubation* lasts from one day to one week. The period of *invasion*, lasting one or two days, usually sets in suddenly. As a rule, there are *chilly sensations* rather than a true *chill*. Fever (Fig. 4) often reaches 104° or 105° F. on the first day, with pallor and prostration. Frequently there is *vomiting* early in the course of the disease, and *convulsions*, especially in young children. The skin is dry and the tongue furred. Even on the first day there may be some dryness of the throat; inspection soon reveals the characteristic *angina*. Cough and catarrhal symptoms are not common.

The *enanthem* is found first, as a rule, upon the *anterior pillars of the fauces*, the *urula*, and the *palate*, possibly extending over the mucous membrane of the cheeks and gums upward into the nose. The enanthem disappears much in the same way as it appears, by desquamation, leaving a coating that

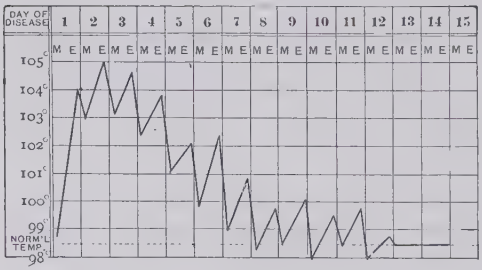
Fig. 3.



Initial fever. Eruption.

Temperature in measles.

Fig. 4.



Eruption.

Temperature in scarlet fever.

"A comparison of the temperature in Scarlet Fever and Measles."

may be confused with that of diphtheria. Usually the enanthem is at its height when the exanthem appears.

The *exanthem* appears on the first or second day, usually *first on the neck, chest, and back*, especially in the *region of the clavicles*, and may spread over the body within forty-eight hours. The *mouth is usually spared*. The color of the eruption is lighter than that of measles—a scarlet—which has

given the name to the disease. The eruption disappears from the face, neck, chest, and body within a week—four to six days—with *desquamation*, frequently in the form of casts, especially of the hands and feet. The appearance of the swollen papillæ protruding through the white coating of the tongue has given rise to the term, "*strawberry*" *tongue*. This may be found in other conditions. The sore throat varies greatly in intensity in different cases.

Scarlet fever—complications: *Nephritis* is the most important complication, and is much more frequent in some epidemics than in others. *Albuminuria* early in the disease may be due to the accompanying fever; but later, from the second to the fourth week, may indicate acute nephritis, which causes *œdema*, especially puffiness of the eyes, *nervous symptoms*, neuralgia, headache, vertigo, insomnia, convulsions, coma, through the effect of irritant products upon the nervous system. *Affection of the ear* is common, and may extend to cause meningitis. *Joint-affections* are sometimes present, probably through secondary infection.

Scarlet fever—forms: The symptoms are sometimes very light and the course of the disease short, constituting the *abortive form* of scarlet fever. The eruption of scarlet fever sometimes remains *localized*, being found only in the face (Braun, Lemoine). Such cases may pass unrecognized and convey the disease. Sometimes the poison is so intense as to take life during the period of invasion, the *fulminant* form; or the symptoms may be exceedingly severe, the *malignant* form. The angina may assume special prominence, the *anginose* form.

Scarlet fever—diagnosis: The presence of an *epidemic*, the history of *exposure*, and of absence of *previous attack* may aid in the individual case.

The *sudden onset* of the disease, often with *vomiting*, one day to one week after exposure; the peculiar *angina;* the characteristic *eruption* on the first or second day; the "*strawberry*" *tongue*, later the *desquamation*, "casts," especially of the hands and feet, and the *complications* on the part of the *kidneys*, *ear*, and *joints*, mark the disease.

Scarlet fever should be differentiated especially from diph-

theria, measles, rubella, septicæmia, acute exfoliating dermatitis, and drug-rashes (belladonna, quinine, iodide of potassium).

Scarlet fever—prognosis: The prognosis *varies greatly in different epidemics*, 3 per cent. (Hirsch) to 90 per cent. (Johannessen). A mortality of 10 to 13 per cent. is considered normal, although in private practice the rate is not so high.

The prognosis in the individual case depends upon the nature of the prevailing epidemic, the character of the infection, and the existing complications. Fulminant and malignant forms always give a grave prognosis. Recovery from nephritis is the rule in scarlet fever. Persistent anuria is ominous. Early in the course of scarlet fever severe nervous symptoms would point to a bad prognosis. Should complications cause the eruption to disappear, to be "driven in," the mortality is increased. An unusually high or low temperature is to be looked upon with suspicion. Other complications that increase the danger of scarlet fever are: severe inflammations about the neck, phlegmonous processes, œdema of the glottis, pneumonia, pleurisy, peritonitis, endocarditis, pericarditis, and meningitis.

Prophylaxis: *Isolation* of the patient should be absolute. Scarlet fever may be conveyed by contact, either direct or indirect, through third persons, clothing, dishes (fomites). Susceptible children should be sent away from a house in which there is scarlet fever. Children who are nursing women sick with scarlet fever rarely contract the disease, and then usually only in a mild form. In all cases quarantine should be continued until desquamation is complete. A collective investigation in English hospitals showed the minimum duration of the infectious period of scarlet fever to be eight and the maximum thirteen weeks. The sick-room should be kept well ventilated, and the patient should be bathed frequently. After recovery is complete the patient should receive a full-length bath and a change of clothing. The sick-room and all articles with which the patient came in contact must be disinfected or destroyed by fire. The nasal mucous membrane should be thoroughly cleansed before the case is discharged.

Gonzales reports good results from the prophylactic use of

sodium sulphocarbolate, where isolation was incomplete or not practised, and claims to have prevented contagion in seventeen families, protecting one hundred and thirty-nine children exposed to scarlet fever.

Scarlet fever—treatment: Good results have been secured by the injection of the blood of recent convalescents; but such treatment is not generally practicable.

The patient should be placed under good hygienic surroundings, in a room where thorough ventilation may be secured and an equable temperature maintained, 65° to 70° F. at the head of the bed. The patient must be kept abed. The best single article of diet is milk. The meat soups afford an agreeable change. Acidulated, mineral, or plain pure water should be offered at regular intervals. A daily full-length warm bath contributes to both cleanliness and comfort. Temperature over 103° F. calls for the application of cold, sponging with cold water, the use of the cold pack, or the cold bath. It is more comfortable to the patient not to have the bath too cold; or, to have the temperature of the water reduced gradually after entering the tub. The bath may be substituted by antipyrin, or more safely by lactophenin or phenacetin, which may also relieve the nervous symptoms, especially headache. Itching of the skin is relieved by the application of lanolin, cocoa-butter, or lard, which should be renewed after each bath.

Mild throat symptoms may not demand treatment; more severe symptoms on the part of the throat call for the application of cold to the neck or the inhalation of steam and the use of antiseptic solutions. In general the local treatment of the throat symptoms in scarlet fever is the same as in diphtheria. When the inflammation extends to the middle ear puncture of the drum-membrane may become necessary.

Turpentine, hypodermatically or internally, is recommended for the prevention of nephritis in scarlet fever. Fauva found the injections to be perfectly harmless. Children may receive one gramme; adults as much as three grammes. Two or three injections are usually sufficient. The digestive organs must be watched, and if necessary the turpentine may be suspended a

couple of days and salines given. To prevent local irritation, sodium bicarbonate may be added to the turpentine.

The treatment of *nephritis* will be considered under acute nephritis.

Cardiac weakness may call for heart-stimulants: alcohol, digitalis, nitroglycerin.

SMALLPOX (Variola).

Definition: An acute infectious disease, characterized by sudden onset with chills, headache, pains in the lumbar and sacral regions, sweating, vomiting, epigastric tenderness, a typical temperature, and peculiar eruption.

History: The disease was probably recognized long before the time of Christ, in India, China, and Central Africa. Smallpox first reached America (the West Indies) in 1597, and the United States (Boston) in 1649.

Etiology: Numerous micro-organisms have been described. Many observers believe the disease to be due to sporozoa. Others believe that the specific cause of smallpox has not been isolated because the microscope is not capable of sufficient magnification to detect the micro-organism.

The *cause* is in the *skin*, as is evidenced by inoculation; and in the *blood*, as suggested by infection of the fœtus and as proven by inoculation-experiments. The disease shows a preference for the cold season.

Smallpox was formerly considered a disease of childhood. Since protection by vaccination has become general smallpox has become so rare that children are seldom exposed to the infection. All ages are susceptible to the disease, with the possible exception of early infancy. Second attacks are rare, as are also attacks after vaccination.

Smallpox—symptoms: *Incubation* lasts eight to fourteen days, usually ten to twelve days. There are no symptoms during incubation, except possibly some malaise late in the period.

The period of *invasion*, lasting two or three days or longer, usually sets in suddenly and violently, with *chill*, rigors, followed by *fever*, rising often to 103° or 104° F. on the first day,

and possibly 105° to 107° F. on the second or third day. The pulse may reach 100–130; in children 160. Prostration is marked.

There are thirst, loss of appetite, often constipation. The tongue is coated and the breath offensive. Some claim that the odor of the breath at this time is characteristic. Very frequently there is *gastric irritation*, sometimes accompanied by epigastric tenderness. Among the nervous symptoms are *headache*, which is almost always present in greater or less degree; delirium, especially when the temperature is high; coma, convulsions, especially in children, and *pain in the back*, especially in females. *Headache*, *pain in the loins*, and *gastric irritation* usually continue from the onset of the disease until the eruption appears.

The *urine* is diminished in quantity, there is a diminution of the chlorides, and in severe cases albumin may be present. A large quantity of albumin in the urine, if not due to chronic disease of the kidney, would probably point to the malignant type of smallpox.

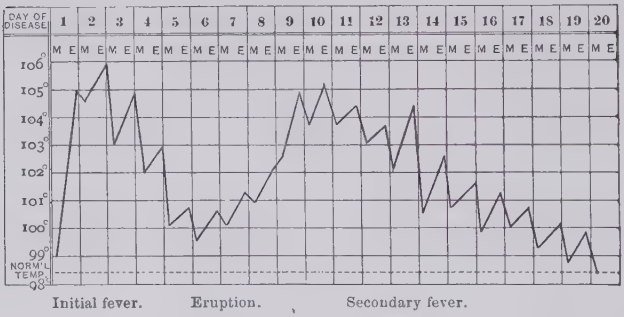
The *spleen* may be enlarged in unmodified smallpox.

Often on the second day of the invasion there is an initial eruption, a *roseola*, lasting not longer than two days, which has been variously described as presenting the appearance of erythema, scarlet fever, and measles. Cases in which this eruption is marked have been observed to run a milder course. Sometimes an initial eruption appears in one of Simon's triangles as *petechiæ*. (Simon's "triangles" are in the groin, hypogastric region, inner surface of thigh, axilla, and inner surface of arm.) This eruption is found most frequently in a triangle, the apex of which is at the knees, and the base on a line extending transversely across the body at the level of the umbilicus. Petechiæ have no diagnostic import.

The *eruption* appears usually on the third day, first on the forehead and temples, near the margin of the scalp, and on the wrists. The eruption shows preference for the cutaneous and mucous surfaces exposed to the atmosphere, spreading rapidly to the scalp, ears, forearms, hands, and to the body and lower extremities in twenty-four hours. At first the eruption appears as little red points, *macules*, which be-

come indurated *papules* in twenty-four hours. The papules feel like shot in the skin. At first discrete, the papules become confluent as they increase in number. About the fifth day of the disease, after the eruption has lasted some three days, the papules which appeared first, will contain serum, at first clear (vesicles), becoming cloudy and milky (pustules), by the fourth or fifth day. The *vesicles* become *umbilicated*. By the sixth day the contents of the vesicles have become distinctly purulent (*pustules*). The vesicles and

FIG. 5.



Temperature in smallpox.

pustules appear *first upon the face* and extend in the order of appearance of the rash over the body and extremities.

With the exanthem there appears an *eruption upon the mucous membranes that are exposed to the external air*, especially the mucous membrane of the *mouth, nose, and pharynx*, sometimes in the vagina, rectum, and urethra.

The *temperature*, which often reaches 106° F., usually *falls when the eruption appears*, but may continue until the third or fourth day of the eruption (Welch). The temperature then falls, to become normal or even subnormal, usually within twelve to eighteen hours. At the same time there is a reduction in the pulse, respiration, and symptoms of gastric irritation. With the *pustular stage* the *temperature again rises*, reaching 102°, frequently 104°, rarely 106° or 107° F., with morning remissions. This secondary fever (Fig. 5) is a

part of the disease, but later shows, as a rule, the "streptococcus-curve" of sepsis (see Septicæmia). During this stage there may be disturbances of the cerebral functions, particularly *delirium* in various degrees.

Desiccation begins on about the eleventh or twelfth day of the eruption, with improvement in all the symptoms. With the drying up of the pustules there is often considerable *itching*, and frequently it becomes necessary to restrain the patients, especially children, from scratching and thus producing unsightly scars. The process of desiccation requires three or four weeks. During this time the fever disappears by lysis.

Smallpox—complications: The *skin* may show multiple abscesses, erysipelas, boils, bedsores, pigmentation from dermatitis, acne pustulosa, and swelling of the hands and feet. The *eyelids* show œdema, possibly with sloughing, and sometimes contain abscesses. Upon the part of the *eyes* there may be conjunctivitis, pustules, keratitis. The chief *ear* complication is deafness, partial or complete. The following are the more common complications on the part of the various organs. The *respiratory organs:* inflammation of the nasal mucous membrane, epistaxis, laryngitis, tracheo-bronchitis, pneumonia, pleuritis. The *circulatory organs:* pericarditis, endocarditis, myocarditis, hemorrhage, venous thrombosis. The *digestive organs:* glossitis, stomatitis, hæmatemesis, diarrhœa, colitis, peritonitis. The *urinary organs:* albuminuria, hæmaturia, acute nephritis, cystitis. The *nervous system:* delirium, meningitis, acute mania, paraplegia, peripheral neuritis, disseminated spinal scleroses, epilepsy, anterior poliomyelitis. The *genital organs:* phimosis, from œdema of the prepuce, orchitis, ovaritis. The most important complication is *secondary infection* (see Septicæmia).

Smallpox—diagnosis: The prevalence of an *epidemic*, the history of a *previous attack*, *inoculation*, or *vaccination*, are points that aid in diagnosis. The *onset* of the disease *suddenly*, with *chills* or rigor, followed by *fever*, *headache*, *pain in the back*, *epigastric tenderness*, and *vomiting*, is suggestive. The appearance of the *eruption* on the third day, first upon the upper part of the *face*, extending rapidly over the body,

changing from *macules* and *papules* to *vesicles*, which are *umbilicated* and later become *pustules*, stamps the disease.

The *differential diagnosis* has to do chiefly with measles, scarlet fever, typhus fever, lumbago, simple fever, syphilis, chicken-pox, erysipelas, drug-eruptions, ptomaine-poisoning, herpes, glanders, acne pustulosa, pemphigus, acute rheumatism, meningitis, malignant or ulcerative endocarditis with erythematous or purpuric rash, and cerebro-spinal fever.

The **prognosis** of smallpox depends largely on the *protection* of the individual by previous attacks of the disease, inoculation, or *vaccination;* the *form of the disease*, hemorrhagic, purpuric, confluent, discrete, abortive (varioloid); the *surroundings* of the patient regarding hygiene, and the *complications* that may arise in the individual case.

The mortality is about 50 per cent. among the unvaccinated, 26 per cent. among the badly vaccinated, and only 2.3 per cent. among the efficiently vaccinated (Moore). Infants and age show a large mortality. The prognosis is not good among drunkards.

Prophylaxis: *Vaccination* is most important. Suspected cases should be quarantined eighteen days. Smallpox patients should be *isolated* until all the scabs and scales have fallen off. They should then be sponged with a solution of bichloride of mercury, 1 : 2000, and given a full-length bath and change of clothing, after which they may safely come in contact with susceptible individuals.

The room to which the patient with smallpox is confined should first be cleared of all unnecessary articles, pictures, curtains, etc. Precautions must be taken that contagion be not conveyed by the physician, attendants, letters, and dishes (fomites). In case of death the body is wrapped in a sheet soaked with bichloride of mercury, 1 : 1000, placed in a hermetically sealed casket, and should be buried as soon as possible. It would be better still to cremate the body.

In the *disinfection* of the sick-chamber and furniture all articles of little value should be burned. Linen and other things of like nature may be boiled at least half an hour or exposed to a high degree of dry heat. Articles which cannot be subjected to moist or dry heat must be spread out in the

room, which is then thoroughly disinfected with formaldehyde gas, chlorine gas, sulphurous acid gas, mercuric chloride, or thiocamf. The apartments should then be well aired.

Smallpox—treatment: Early vaccination, within the first four days, may lessen the severity of the disease.

Béclerè has reported the successful treatment of sixteen cases of smallpox with injections of the serum of a vaccinated calf. One and a half liter of the serum was injected under the skin of the abdomen, in three doses. All the cases recovered.

Otherwise the treatment is *symptomatic*. The patient should be kept in bed, in a well-ventilated room with a temperature of 65° F. The *diet* should be light—milk, soups, gruels—and the patient should be given plenty of pure water to drink. *Pain* calls for opium; *fever* above 103° F. for baths, phenacetin, salipyrin, antipyrin. The *throat symptoms* may require inhalations of steam, antiseptic gargles or sprays. Ice is grateful. The *nervous symptoms* may be met with chloral or Dover's powder. *Pitting* may be limited, before the formation of pustules, by touching the vesicles with pure carbolic acid. Some prefer anointing with oil or vaseline and covering the parts, especially the face, with lint soaked with a solution of bichloride of mercury, 1 : 5000 or 1 : 10,000; a 1 per cent. solution of creolin, or a dilute solution of carbolic acid. The object is to prevent infection of the vesicles, and consequent destruction of tissue.

Varioloid.

Mild cases of smallpox—**varioloid**—appear most frequently in individuals who have received a certain degree of immunity or protection through vaccination, inoculation, or previous attack of smallpox. Varioloid assumes special importance since infection from cases of varioloid may cause unmodified smallpox.

Vaccinia; Vaccination; Cowpox.

Definition: Vaccinia is the name given the disease produced in man by inoculation (vaccination) with cowpox.

History: Vaccination in prophylaxis against smallpox was introduced by Edward Jenner, 1798.

Etiology: As in smallpox, many micro-organisms have been described in vaccinia, but the etiologic relationship of none of them has been definitely established. Kent believes the specific organisms of vaccinia to be a *diplobacillus*. Inoculation of susceptible animals with pure cultures of this organism causes vesicles that may not be distinguished from those produced by vaccine lymph, and the animals so inoculated are immune to the action of vaccine lymph. The vesicles produced by inoculation contain the diplobacilli in large numbers. The supposition that cowpox is a bovine smallpox, as well as the belief that cowpox is identical with horsepox and sheeppox, remains without final proof, although no one will deny the close relationship of these affections. Cowpox will protect man against smallpox, and inoculation with smallpox will protect cattle against cowpox. The immunity against smallpox, in man, begins on the fourth day after vaccination, and is highest by the ninth day, but does not remain complete for life. Revaccination, in order to secure the most perfect immunity, should be repeated as often as it will "take." Virus from the cow or from cases of vaccinia in man may be used. The chief danger from the use of humanized lymph lies in the possibility of syphilitic infection, which may be avoided by the use of virus from a healthy individual, or more absolutely by the use of bovine virus, best in the form of so-called *glycerinated lymph*. Susceptibility to vaccination is universal, premising no previous attack of smallpox or vaccinia. Failure of the virus to "take" calls for another attempt.

Vaccination—method: The virus should be rubbed into abrasions of the skin, made most conveniently by scarification or incision, which should cause the exudation of lymph through exposure of the superficial lymphatics, and little or no flow of blood. The point of selection, as a rule, is the left arm, near the insertion of the deltoid muscle, but usually there is no objection to vaccinating on the leg or some other part of the body.

VARICELLA (Chickenpox; Waterpox).

Definition: An acute infection of childhood, occurring especially from two to six years of age, characterized by an eruption of vesicles, waterpox. Chickenpox is a common name for the disease, but varicella is not known to have any connection with chickens.

Etiology: The specific agent of infection, probably a microorganism, has not been demonstrated. Usually one attack confers immunity. So far as we know, there is no relationship between variola (smallpox) and varicella (chickenpox). Varicella shows a decided preference for children, especially under ten years, and very rarely attacks adults.

Varicella—symptomatology: *Incubation* lasts eight to seventeen days. The *invasion* is marked by fever with possibly a chill, vomiting, and pain in the back and legs, rarely convulsions. The *eruption*, first seen on the trunk, back, or chest, *develops in a day.* At first the eruption is papular, the little red papules becoming vesicles in a few hours. The vesicles may show umbilication, though this is usually not the case. Within two days the vesicles become purulent, and a day or two later the eruption dries up, the crusts falling off, to leave as a rule no scar. The eruption appears in successive crops.

Varicella—diagnosis: The symptoms are usually much milder than in smallpox. Prodromata are rare in varicella. The *pocks* rarely present a feeling as of shot under the skin, such as is found in smallpox; they appear especially upon the trunk, rarely become confluent, do not present so great infiltration around them, and are apparently more superficial. Prodromal rashes are more common in smallpox than in chickenpox. Above all, varicella occurs in childhood, sometimes as an epidemic, but *never* appears as an epidemic among adults. Further, varicella shows no respect for vaccination.

Prognosis: Good. Complications do not often occur.

Varicella—treatment: The patient should be confined to the house, if not to bed. The diet should be light. As a rule little or no treatment is required. Irritation of the skin

may call for the application of cocoa-butter or a dilute solution of carbolic acid.

DIPHTHERIA.

Definition: An acute infectious disease, caused by the Klebs-Löffler bacillus, characterized by a fibrinous exudate, false membrane, occurring especially upon the mucous membrane of the throat, occasionally upon other mucous membranes and wounds.

Diphtheria—history: The disease was recognized by the older physicians—Aretæus, Galen—but was not dissociated from other forms of sore throat. Early in the nineteenth century diphtheria was recognized by Bretonneau as a separate affection. The bacillus of diphtheria was discovered by Klebs, 1883; isolated, cultivated, and its pathogenesis demonstrated by Löffler, 1884.

Etiology: The specific infectious agent in true diphtheria is the *bacillus diphtheriæ*, commonly known as the Klebs-Löffler bacillus. Most cases of diphtheria show mixed infection with streptococci and staphylococci. Diphtheria is endemic in cities. Children from two to fifteen years of age are especially liable to attack.

Diphtheria—symptomatology: *Incubation* two to seven days. The period of *invasion* is announced by slight *chilliness, fever*, pains in the back and limbs. In mild cases the patient may not feel sick enough to keep abed. During the *first day* the *temperature rises* to 102°–103° F., possibly 104° F. Sometimes in childhood there are convulsions.

In *pharyngeal diphtheria*, membranous croup, there is some inflammation about the tonsils, palate, and pharynx, constituting the *catarrhal stage*. The patient complains of *dryness, burning, and constriction of the throat, and difficulty in swallowing*. Soon the fibrinous exudate appears as a *false membrane*, usually first upon the tonsils, extending by the third day to the fauces and uvula, possibly later to the posterior wall of the pharynx. The color of the membrane changes from a grayish-white to a dirty gray, possibly to a yellowish-white. At first the false membrane may be readily detached, but later the mucous membrane is so involved that the false membrane

may be removed only with difficulty, leaving a bleeding surface, which soon again is covered with fresh exudate. Later the false membrane may be readily removed, after mixed infection. *Convalescence* usually begins about the seventh to the tenth day. Occasionally the disease lasts longer. Jessen reports a case lasting for five months, in which there were virulent diphtheria bacilli. Such cases are rare.

The diphtheritic process may involve the nasal mucous membrane, *nasal diphtheria;* or the false membrane may extend to the larynx, *laryngeal diphtheria;* or to the bronchi, œsophagus, or Eustachian tube. Cases of diphtheria may appear primarily in the larynx or nose.

Diphtheria—complications: One of the most important complications is post-diphtheritic *paralysis*, of toxic origin, affecting especially the *palate*, possibly the epiglottis or larynx, or the constrictors of the pharynx, interfering with deglutition; sometimes involving the *eye* (strabismus, ptosis, and alterations of accommodation); sometimes affecting the face, or *extremities, especially the legs*, frequently with *loss of the knee-jerk.*

Albuminuria is found in all severe cases. Albumin in the urine in considerable quantity, with casts, would indicate nephritis. The *heart* shows both functional and organic derangement, tachycardia, bradycardia, pericarditis, endocarditis, valve-lesions, and heart-failure. Capillary bronchitis and broncho-pneumonia are found in the more severe cases.

Diphtheria—diagnosis: The presence of an *epidemic*, and the *history* of *exposure* to infection and absence of *previous attack*, are valuable aids in some cases. A *false membrane* may be present in some recess, as in the nose, and not be visible. Mild cases sometimes escape recognition until they spread infection and the disease appears in a more severe form. The demonstration of the *bacillus diphtheriæ* is of the greatest value in diagnosis, since it differentiates diphtheria from pseudo-diphtheria and reveals the character of mild cases of diphtheria, which might otherwise pass unrecognized and become foci of infection. The value of the bacteriological examination in diphtheria is well indicated in the emphatic statement by Osler, that "Where a bacteriological

examination cannot be made, the practitioner must regard as suspicious all forms of throat affections in children, and carry out measures of isolation and disinfection." The occurrence of *albuminuria with casts* points to diphtheria.

Examination for the bacillus diphtheriæ: The bacillus grows upon various media—milk, potato, alkaline bouillon, nutrient gelatin, glycerin-agar, etc. The growth is most rapid upon the blood-serum mixture recommended by Löffler: blood-serum, three parts; bouillon, one part; to which are added peptone and grape-sugar, each 1 per cent., and sodium chlorid $\frac{1}{2}$ per cent. After sterilization the mixture is solidified at a low temperature.

A test-tube containing Löffler's blood-serum mixture is inoculated from a swabbing of the throat, or a piece of the false membrane, and placed in the incubating-oven at a temperature of about 35° C. for twenty-four hours. If the case be one of diphtheria, large, moist, grayish-white, elevated colonies of the bacillus diphtheriæ will be present, usually without sufficient development of other micro-organisms to interfere with the examination.

From one of the colonies a slide is prepared and stained with Löffler's alkaline methylene-blue solution: saturated solution of methylene-blue, 30 c.c.; solution of caustic potash, 1 : 10,000, 100 c.c. Or a good double stain may be secured by using the modified Weigert's fibrin stain and picrocarmine, recommended by Welch and Abbott.

The specimen should be examined under the microscope with an amplification of a thousand diameters or more.

The bacillus diphtheriæ is non-motile, grows either in the presence or absence of oxygen (facultative anaërobic); it does not liquefy gelatin and does not form spores.

The pseudo-diphtheria bacillus is often found associated with the true bacillus diphtheriæ, which it may closely resemble. Sometimes the pseudo-diphtheria bacillus may be differentiated only by the lack of virulence.

Diphtheria—prognosis: Usually good in private practice in cases that are seen early. A greater mortality occurs in hospital practice, since many cases are far advanced when they apply for treatment. Some epidemics give a higher mortality

than others. In general the prognosis is very much better since the introduction of the antitoxin treatment. Koenig and Moxter used antitoxin successfully in an infant five days old.

Prophylaxis: For the protection of the community, *a patient with diphtheria should be isolated* as long as cultures reveal the presence of the bacillus diphtheriæ. Thus it may be necessary to prolong isolation far into convalescence. This applies to mild as well as severe cases. Infection may be conveyed by third parties—fomites. When the disease has terminated the sick-room and all articles that have come in contact with the patient should be sterilized. In cases exposed to infection an injection of antitoxin may prevent the disease. Such immunity lasts from two to four weeks. Infants at the breast rarely contract diphtheria from an infected nurse. In such cases Schmid and Pflanz have demonstrated, in Escherich's clinic, that the nurse's milk contains diphtheria antitoxin. If for any good reason the prophylactic dose of antitoxin cannot be given subcutaneously to nursing infants, it may be administered internally pure or mixed with eggs or milk. When given internally the prophylactic action of antitoxin is not so reliable as after subcutaneous injection in infants, and is of no value in adults. The internal use of antitoxin cannot be depended upon in the treatment of diphtheria. The use of antiseptic sprays, in cases that have been exposed to diphtheria, is of value.

Diphtheria—treatment: The diet should be light, consisting chiefly of milk, eggs, carbohydrates, butter, and light meats.

Antitoxin should be used as early as possible in all cases, after a diagnosis of diphtheria has been made. In doubtful cases it is better to make the injection of serum before a bacteriological examination is made, if such an examination cannot be made promptly, especially in cases not seen until the third or fourth day of the disease. Patients over two years of age may receive from 1500 to 2000 units; infants under two years, 1000 to 1500.[1] The serum may be obtained

[1] An antitoxic unit (Behring-Ehrlich) is ten times the amount of antitoxin which, when mixed with ten times the minimum fatal dose of toxin

either in the liquid or solid form. The solid antitoxin is soluble in ten parts of water, and is of such strength that one gramme represents 5000 immunity units. If no improvement follows the first injection of antitoxin, the dose should be repeated in eighteen to twenty-four hours, and again after a similar interval, if necessary. Antitoxin has complete control over the infection by the bacillus diphtheriæ, but not over the secondary infection by other micro-organisms (see Septicæmia).

Local applications should be made of the subsulphate of iron. Some advise the use of the tersulphate or the perchloride of iron, fuming hydrochloric acid, carbolic acid, bichloride of mercury, creosote, or creolin. The surface is best touched or painted with the remedy, with a cotton-wrapped sound, care being taken that none of the fluid drops into the larynx.

Comfort is sometimes secured by the inhalation of steam. The nasal passages may be kept moist with yellow oxide of mercury ointment and vaselin, 1 : 6, introduced several times a day. Antiseptic sprays may be used; but, as a rule, these accomplish more in the prevention than in the cure of diphtheria.

The *toxic symptoms* are best met with alcohol. A weak heart may be supported with digitalis, best in the form of the fresh infusion. Quicker results are secured with nitroglycerin. Severe cases may demand the use of camphor, ether, alcohol, or musk subcutaneously. In all cases, especially in adults, the patient should be kept abed, that undue strain may not be thrown upon the heart. Paralysis calls for the use of electricity and strychnin. Severe dyspnœa, indicating obstruction of the larynx, may demand intubation or tracheotomy.

Complications should receive proper attention.

for a 250-gramme guinea-pig, and injected subcutaneously, will neutralize the poisonous effect of the toxin and permit the test-animal to remain apparently unaffected. The guinea-pig must not vary in weight over 15 grammes from 250 grammes, and on the seventh day after the injection must be alive and within 20 grammes of the original weight.

Croup.

True croup is characterized by a peculiar crowing inspiration, due to the presence of a false membrane in the larynx.

In true membranous croup the most common cause is the bacillus diphtheriæ (see Diphtheria). In other cases the specific etiological factor is some other micro-organism, most frequently the streptococcus, especially in cases that are secondary to the acute infectious diseases, measles, scarlet fever, whooping-cough, rötheln, smallpox, typhoid fever, less frequently after simple catarrh. Exceptionally cases may depend upon mechanical or chemical irritation, excessive heat and dryness, ammonia, chlorine, bromine, and the fuming mineral acids.

As a rule croup occurs in children from two to seven years of age, although infancy is not exempt, and exceptionally the disease appears later in life.

False Croup—Laryngismus Stridulus.

False croup is characterized by a peculiar crowing inspiration, due to a laryngeal spasm.

There is no false membrane in the larynx, such as is found in true croup. Sometimes there is a light catarrh of the larynx. The disease is found most frequently between six months and five years, especially in children that are confined in badly ventilated apartments. The attacks depend upon spasm of the adductors of the cords.

QUINSY (Epidemic Tonsilitis; Suppurative Tonsilitis; Parenchymatous Tonsilitis).

Definition: An acute infection of the fauces, pharynx, and tonsils, probably contagious, that tends to go on to suppuration.

The disease prefers fall and winter, and is found most frequently in adolescence, from fifteen to thirty years.

Symptomatology: The symptoms of quinsy are marked by their *severity*. The disease comes on with *chill and fever*, 103°–105° F.; pulse 110–130. There are anorexia, some-

times nausea, pain in the back and limbs, headache, and extreme prostration. The throat is sore and dry. There is *difficulty in swallowing.* One or both tonsils may be affected. The *tonsils are enlarged* and œdematous, sometimes to such a degree as to meet in the median line, or one tonsil may be enlarged so as to extend beyond the median line. The swelling may be detected on the outside of the neck. Deglutition is painful. The tonsils are tender and soon show evidence of the presence of pus. *Pus* is recognized early by palpation.

Extreme enlargement of the tonsil and surrounding tissue impairs hearing by blocking the Eustachian tube, and interferes with the use of the voice.

Prognosis: Good. Death may be caused by the abscess bursting and inundating the larynx, or rarely by opening into the internal carotid artery. But recovery is the rule. Some individuals seem peculiarly liable to repeated attacks.

Quinsy—treatment: Moist heat should be applied in the form of poultices, hot water, or steam. Pain may be controlled best with Dover's powder, internally. Suppuration calls for evacuation of the pus.

TUBERCULOSIS.

Definition: An infection due to the bacillus tuberculosis, characterized by the formation of tubercles (nodules).

Tuberculosis—history: The disease was recognized as a *suppuration* by the older clinicians. Later, after the birth of anatomy in the sixteenth century, *nodules* were observed by the anatomists. Early in the present century Bayle and Laennec ascribed the disease to the deposit of *tubercle*, which they showed to be a specific product, independent of ordinary inflammation. Villemin (1865) produced tuberculosis experimentally by inoculation with tuberculous sputum, and declared the disease to be caused by a *virus*. Koch (1882) isolated the virus as the *tubercle bacillus*.

Tuberculosis—etiology: The specific etiological factor is the *bacillus tuberculosis*, which gains entrance to the body through the inspired air (*tuberculosis pulmonum*); or the food, especially the milk and meat (*tuberculosis intestinalis*); or by direct

infection of wounds (*lichen tubercle*); and possibly through heredity (*congenital tuberculosis*).

Cornet believed tuberculosis to be disseminated chiefly through the dried sputum. Flügge has shown that the disease is most frequently spread through the agency of minute droplets of fluid containing bacilli, ejected during coughing, sneezing, etc.

Secondary infection by streptococci, in cases of tuberculosis, intensifies the virulence of the toxins to a higher degree than would be present in either infection alone.

Among the factors *predisposing* the individual to infection, environment—*exposure to infection*—is the most important. Bad hygienic surroundings, especially crowding, exclusion from fresh air and sunlight, a sedentary life, and exposure to dust render the individual more liable to infection. Certain diseases, especially bronchitis, measles, whooping-cough, influenza, diabetes, chronic nephritis, cirrhosis of the liver, chronic heart-disease, arterio-sclerosis, aneurism of the aorta, and possibly above all *trauma*, prepare the body for infection by the tubercle bacillus.

Affection of the *tonsil* may be primary or secondary, in the latter case being due to infection carried by the return-flow of lymph (Schlesinger). Such an explanation is supported in some cases by finding the deposit at the base of the tonsils, away from the crypts. Tubercular *stomatitis*, more often tubercular *ulcer of the intestine*, depends especially upon the swallowing of tuberculous sputum or the ingestion of food (milk) containing the tubercle bacillus.

Laryngeal tuberculosis is usually secondary to involvement of the lungs. Cases of primary tuberculosis of the larynx are occasionally reported. Sometimes tuberculosis presents the general appearance of *pneumonia*.

Tuberculosis of the *kidney* is not infrequent.

Lichen tubercles are caused most frequently by scratching with contaminated hands.

Tuberculosis of the *inguinal glands* following circumcision has been reported in a number of cases. Ten such cases were inoculated by one operator, who himself later died of pulmonary tuberculosis (Ware).

Tubercular *myositis* has been reported in some sixteen cases.

A number of investigators have observed branching forms of the tubercle bacillus with club-shaped extremities, resembling the ray fungus. These are believed by some to be degenerative forms, or a reversion to the type of organism from which the tubercle bacillus was originally evolved. Friedreich, Babes, and Levaditi found the branching forms with club-shaped extremities early in the course of experimental tuberculosis. Brons proposes the term "myco-bacterium of tuberculosis" for the tubercle bacillus.

Tuberculosis—symptomatology: Most cases begin with *bronchitis*, manifested by *cough*, at first dry and hacking, occurring especially in the morning and evening upon changing the posture. The *expectoration*, at first absent, becomes abundant; at first mucoid, later muco-purulent, and possibly containing blood. Microscopic examination of the sputum reveals the presence of the *bacillus tuberculosis*, later *elastic tissue*. *Hæmoptysis* usually means tuberculosis.

Frequently the first symptom noticed is *dyspepsia*, often associated with *anæmia*, *chlorosis*, *amenorrhœa*, and *general degradation of health*. These are regarded as symptoms of toxæmia.

Only too often the onset is so insidious as not to cause the patient to seek medical advice until the disease is far advanced.

One of the early symptoms is *shortness of breath upon exertion*. Later there is *dyspnœa*, due to cardiac weakness, sometimes associated with cyanosis. *Pain in the chest* is a common symptom, due to pleurisy, sometimes to neuralgia of the intercostal nerves, caused by toxæmia. The *temperature* at first may be normal or subnormal in the morning; but shows early a rise some time during the day, usually in the afternoon. With the fever there may be *night-sweats*. Later the temperature becomes higher, 103° F., possibly 104° F., with daily remissions of two or three degrees. After secondary invasion by the pyogenic micro-organisms takes place, constituting the period of "*hectic*," the temperature varies from 103° to 105° F. in the evening, but is normal or sub-

normal in the morning, constituting the "*streptococcus-curve*" (see Fig. 1). The night-sweats may be exhausting. The *pulse*, at first corresponding to the temperature, with increased weakness becomes rapid, compressible, and readily influenced by exercise.

Frequently one of the earliest symptoms is *loss of weight*, and in the later stages emaciation is so marked as to have been one of the first recognized signs of the disease. Hence the terms, *phthisis*, consumption (wasting).

Laryngeal tuberculosis will receive separate consideration.

Tuberculosis—physical signs: Physical signs at first are entirely absent. *Inspection* may reveal the characteristic long, narrow chest, and the winged scapulæ, which have been aptly compared to folding doors or the wings of the eagle. The clavicles become prominent. The chest may show deformity.

The *habitus phthisicus*, marked by a long, flat chest, with emaciation and weakness, formerly believed to *predispose* to tuberculosis, is now recognized as evidence of the existence of the disease.

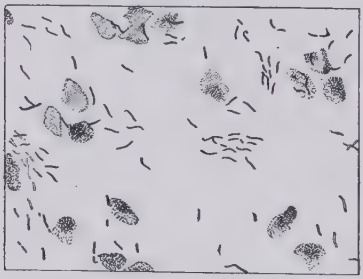
Palpation shows lessened mobility, with defective expansion on one or both sides. With consolidation, vocal fremitus is increased. In cases of pleural exudate the vocal fremitus is diminished or absent. *Percussion* may reveal defective resonance, especially in the region of the clavicle. In advanced cases percussion will show dulness from consolidation, the so-called fibroid change; or a cracked-pot sound may be caused by the presence of cavities. *Auscultation*, as a rule, shows prolonged expiration early in the course of the disease. Later all sorts of râles may be heard.

Tuberculosis—diagnosis: In cases far advanced the *physical signs* leave little doubt as to the character of the disease. Advanced cases often show *elastic tissue* in the sputum; but this may appear in other diseases, especially in abscess or gangrene of the lung, and sometimes is not present even late in tuberculosis. As a rule, an earlier diagnosis may be made by the discovery of the *tubercle bacillus* (Fig. 6).

Examination for the bacillus tuberculosis: Some sputum is collected in a clean vessel. From the specimen a suspicious yellowish or whitish particle is selected, or a film is spread on a

slide or cover-glass with a camel's-hair brush. If the sputum is very tenacious, it may be better to add some caustic soda or potash and precipitate the bacilli with the centrifuge. The film is allowed to *dry*, and is then *fixed* by passing through a flame, specimen side up, three times. The specimen is now ready for the *stain*. Probably the most satisfactory stain is the *Ziehl carbol-fuchsin solution:* fuchsin, 1 c.c.; absolute alcohol, 10 c.c.; carbolic acid crystals, 5 c.c.; distilled water, 100 c.c. The specimen is covered with, or floated upon, this solution, under gentle heat, just sufficient to cause steam

FIG. 6.



Tubercle-bacilli. Sputum of a man suffering from tuberculosis of the lung, spread in a thin layer on a cover-glass and stained with fuchsin and methylene-blue (Ziegler).

to rise, usually *three to five minutes*. *Decolorize* everything but the tubercle bacillus by the use, for about *thirty seconds*, of *acid alcohol:* hydrochloric acid, 1 c.c.; 70 per cent. alcohol, 100 c.c. *Wash* with *absolute alcohol* and then with *water*. *Counterstain* with a saturated aqueous solution of *methylene-blue*. *Wash* off the surplus stain with water, *dry* and *mount*, best in glycerin or balsam. The tubercle bacilli appear red upon a blue background.

Tuberculin test: Still earlier in the course of tuberculosis, before bacilli are thrown off through the sputum or when they are so few in number as to be difficult to find, the diagnosis may be made by a test-injection of one milligramme of Koch's old *tuberculin*, which causes a rise of fever in tuberculosis, but no temperature-reaction in non-tubercular

cases. This method is of especial value, since it discloses tuberculosis not only of the lungs, but anywhere in the body. The cases in which other diseases (actinomycosis, leprosy) have been reported to give the reaction were probably cases in which a coincident tuberculosis in some part of the body was overlooked.

Pronounced agglutination and bactericidal power have been found in the serous fluid from local tuberculous lesions. In non-tubercular cases there is no such reaction.

Tuberculosis—prognosis: The spontaneous cure of tuberculosis is not uncommon. In fully one-third of autopsies (Mossini, 39 per cent., quoted by Osler) upon individuals who have died of some disease other than tuberculosis there is evidence of pre-existing tuberculosis.

In a general way it may be said that two-thirds of mankind have tuberculosis, and that two-sevenths succumb to pulmonary tuberculosis, and fully one-third to tuberculosis in some form, including affections of the intestine, bones, glands, etc.

Much depends upon the environment, especially as regards autoinfection of patients from their own sputum. The symptoms of sepsis are ominous. When treatment is begun before the development of septic symptoms the outlook is not so bad.

Froebelius, in the post-mortem examination of 18,569 *infants*, found the cause of death to be tuberculosis in 416, about 0.4 per cent.

Tuberculosis—prophylaxis: The community as well as the individual should be protected by *destruction of the sputum*, best by fire. Promiscuous expectoration should be absolutely prohibited. Cuspidors must contain water or some antiseptic solution. Crowding, especially in a tuberculous atmosphere, favors contagion, as do also the inhalation of dust and the exclusion of fresh air and sunshine.

It would be better for society if all tuberculous patients could be isolated.

After a case of tuberculosis the sick-room and everything that has come in contact with the patient should be disinfected.

For the protection especially of infants, dairies should be

inspected systematically with reference to the presence in the milk of tubercle bacilli. Tuberculous animals should be killed.

Tuberculosis is found less frequently by far among the *cattle* of Colorado than among the cattle raised in less elevated portions of this country. Gardiner concludes that in a drink of *milk* taken in a city below 2000 feet above the sea-level the risk of tuberculosis intestinalis is about 30 per cent. greater than in an elevated region such as Colorado.

Tuberculosis—treatment: In the way of specific medication, the new *tuberculin* of Koch (T. R.) is of the greatest value, especially in cases of *pure tuberculosis*, but it has *no control over the "sepsis" of phthisis.*[1] The remedy is given hypodermatically, preferably in the back, beginning with 0.001–0.002 mg. and gradually increasing up to 20.0 mg. The tuberculin is usually dissolved in 20 per cent. glycerin, to which 0.6 per cent. sodium chloride has been added. A solution containing $\frac{1}{5\,0\,0}$ mg. of tuberculin to 1.0 c.c. is used at first, the injections being given every day, sometimes every second or third day, beginning with 1.0 c.c. of the solution, or, in very bad cases, 0.5 c.c., and increasing 1.0 c.c. each injection until the dose reaches 10.0 c.c. The solution is then increased in strength and the injections again gradually increased from 1.0 c.c. to 10.0 c.c. Again the solution is increased in strength, and so on until pure tuberculin is used, gradually increasing the intervals between injections as the injections increase in size, so that when the large doses are reached, the patient receives

[1] Besides Koch's new tuberculin, commonly known as T. R., prepared from whole tubercle bacilli pulverized in a mortar, the following have been recommended to address the specific cause of tuberculosis: Tuberculin (Koch), a glycerin extract of the tubercle bacillus, commonly known as Koch's old tuberculin, which is used chiefly for diagnostic purposes; tuberculocidin (Klebs), a modification of tuberculin; antiphthisin (Klebs), practically same as above; tuberculinum purificatum (v. Ruck), practically ditto; purified tuberculin (Whitman), practically ditto, made from the culture-fluid in which the tubercle bacilli have grown; oxytuberculin (Hirschfelder), a 5 per cent. solution of tuberculin saturated with peroxide of hydrogen under protracted heat; V. Ruck's aqueous extract of dead tubercle bacilli.

Serums: Maragliano's serum; antitubercle serum (Paul Paquin); Crandall's immunized serum; Mulford's ass' serum, antituberculin serum; antiphthisic serum (Fisch).

one injection in one or two weeks. The injections should cause no fever, and are of most value when there is no, or but little, fever.

The solutions of tuberculin must be kept where it is cool but not damp; and should they become cloudy they must not be used. This caution is important. For the laryngeal ulcers perhaps nothing is better than the local application of lactic acid.

Fever and *night-sweats*, evidences of the sepsis of phthisis, disappear when the individual remains in the open air day and night. Night-sweats may be controlled with atropine, gr. $\frac{1}{150}$–$\frac{1}{60}$. Aromatic sulphuric acid combined with gallic acid is also highly recommended. *Diarrhœa* may be controlled for a time with bismuth, best in combination with Dover's powder, or by enemata of starch and laudanum, or acetate of lead and opium in pill, or by tannalbin or tannigen. *Hæmoptysis*, when the hemorrhage is from the lungs, may be relieved by morphine and atropine, at first hypodermatically, later per os, with rest in bed and the application of an ice-bag over the heart. Among other remedies recommended for hæmoptysis are: ergotin, sclerotinic acid, aconite, aromatic sulphuric acid, tannic acid, lead, and gallic acid.

For the relief of the *septic symptoms*, aside from life in the open air, probably nothing equals the aromatic oils containing sulphur. Of these the oil of garlic is especially beneficial, but its use is seldom practicable because of the odor.

Intrapulmonary injection with iodoform in oil has been suggested by the success attending the use of iodoform in surgical tuberculosis, as of the joints, and deserves a further trial.

Alexander recommends campor subcutaneously. The remedy is antihydratic, antipyretic, and a cardiac stimulant, and lessens the suppuration and cough.

Sanitaria are of value in the management of tuberculosis chiefly in so far as such institutions may secure the proper climatic, hygienic, and dietetic treatment of cases.

Frequently good results may be obtained by the use of cinnamic acid, or, better, the cinnamate of sodium, given by intravenous injection.

Nebulization of the various essential oils has been recommended. These remedies may exert a beneficial effect, especially upon the sepsis of phthisis, but are not a satisfactory substitute for the open-air treatment.

Recovery from *tubercular meningitis* has followed lumbar puncture.

In *tubercular laryngitis* pain may be relieved by insufflations of orthoform, gr. v, or by painting the surface with a 10 per cent. solution of the hydrochlorate of orthoform (Naumayer).

Tubercular pleuritis which does not show a tendency to undergo resolution promptly may demand thoracentesis and possibly the resection of one or more ribs.

LEPROSY (Lepra; Elephantiasis Græcorum; Aussatz (German)).

Definition: A chronic infectious disease, due to the *bacillus leprœ*, characterized by changes in the skin (*tubercular leprosy*), and in the nerves (*anæsthetic leprosy*), and also in the bones and other tissues.

History: Probably reference was made to leprosy by Moses. The disease was described in detail by Celsus, 25 A. D. History shows the *gradual extension* of leprosy *westward*, from Egypt to the Orient, India, Persia; to Greece, Italy, later to Spain, France, and Germany; to England and Scotland in the tenth century. It was spread all over Europe by the Crusades in the eleventh and twelfth centuries, to be brought under control by *segregation* in the fifteenth to the seventeenth century.

Leprosy is *endemic* in northern and eastern Africa, Madagascar, Arabia, Persia, India, China and Japan, Norway and Sweden, Italy, Greece, France, Spain, and the islands of the Indian and Pacific Oceans. *The disease is found* in Central and South America, Mexico, the West Indies, Hawaii, Australia, and New Zealand, and also in New Brunswick, Canada. *In the United States* most cases occur in Louisiana and California, occasionally in Florida, New York, Ohio, Pennsylvania, Minnesota, Missouri, North and South Carolina, and in Texas.

Etiology: The *bacillus leprœ* is generally accepted as the specific cause of leprosy, although this has not been demonstrated by the production of the disease in man through inoculation with a pure culture of the bacillus.

The bacillus lepræ bears a marked resemblance to the tubercle bacillus, from which it may be differentiated by bacteriological methods, especially by its affinity for acid stains, such as eosin and acid fuchsin.

Leprosy may be communicated through inoculation. The disease is probably not contagious except upon close contact. As a rule, cases do not present open ulcers; and when there are ulcers the superficial bacilli are usually dead. Hereditary transmission has not been proven.

Kaposi has reported a case in which a leproma developed where an individual had been bitten upon the finger by a mosquito. Sommer has observed that leprosy is more frequent where mosquitoes occur in large numbers.

Leprosy—symptomatology: The period of *incubation* has been stated to be from a few weeks to as long as twenty or even forty years, probably most frequently from *three to five years.*

Among the *premonitory symptoms* are irregular fever, malaise, anorexia, dyspepsia, epistaxis, dryness of the nasal passages and respiratory tract, vertigo, headaches, neuralgias, rheumatic pains, articular pains, exaggerated functions of the kidneys and sweat-glands, anxiety, pruritus, and hyperæsthesia of the skin.

Erythematous eruptions may appear in various parts of the body, constituting the *macular stage.* Bullæ may appear over the articulations of the fingers and toes, knees, elbows, wrists, and ankles. The appearance of *nodules*, especially upon the forehead, eyelids, nose, lips, chin, cheeks, and ears, constitutes the so-called *tubercular leprosy.* The nerve-trunks are thickened.

Anæsthetic leprosy may start as an ulcer, appearing usually as the result of numerous bullæ re-forming in the same locality. Patches of anæsthesia, sometimes hyperæsthesia, may appear in various parts of the body.

Anæsthesia of the little finger is said to be one of the most

constant symptoms, often appearing before other lesions in the hand. The tendon reflexes are exaggerated. Later there are marked trophic changes, atrophies, paralyses, etc.

Usually the disease appears first as the tubercular form of leprosy, sometimes as the anæsthetic form, and later takes on the symptoms of the other variety.

Leprosy—diagnosis: Usually the history and symptoms are sufficient to make the diagnosis. In case of doubt, the body, including the suspicious maculæ, may be rubbed with fuchsin methyl-violet in powder, then covered with absorbent cotton, and perspiration caused by the injection of pilocarpin: the sound skin will be colored, while the leprous spots, which do not perspire, will not be stained (Baelz). Or the tissue may be searched for the bacillus lepræ.

Leprosy should be *differentiated* from morphœa, syphilis, iodism, sarcoma, molluscum fibrosum, lichen planus, dysidrosis, and Morvan's disease (if this be not, indeed, a variety of leprosy, or leprosy a causative factor of Morvan's disease).

Leprosy—prognosis: The average duration of life, about eight years, is greatly exceeded in some cases.

Prophylaxis: *Segregation* will prevent spread of the disease. In all cases absolute cleanliness should be observed, including destruction of all excreta and the protection of open sores. The patient should use individual utensils and occupy a separate sleeping-apartment.

Leprosy—treatment: Probably the remedy of most value is gynocardia (chaulmoogra) oil, Gynocardia odorata, given internally, beginning with gtt. ij in capsule or milk and increased to tolerance, usually about two drachm doses, half an ounce a day.

Among many other remedies which have been highly recommended may be mentioned gurgun oil (dipterocarpus turbinatus), ℥v-x internally with lime-water; ichthyol, internally and externally; pyrogallic acid and resorcin, externally; salol and salicylate of sodium, internally; morphine and oil, hypodermatically; potassium chlorate, internally in large doses (gr. 180-380 per day, Dyer); oxygenated muriate of potassium; the bites of venomous snakes; the serum obtained from leprous lesions; the use of tonics, stimulants, and pallia-

tive measures. Various baths are recommended. In many cases an appeal must be made to surgery.

SYPHILIS (Pox; Lues Venerea).

Definition: A chronic infectious disease, transmitted through heredity (*congenital syphilis*) or through inoculation (*acquired syphilis*).

In *acquired syphilis*, a sore at the site of inoculation, after an incubation of two or four weeks, constitutes the *primary lesion*. Two or three months later the *secondary lesions* develop: affections of the skin and mucous membranes, sore throat, cutaneous eruptions, and condylomata. The *third stage* of the disease develops after a period of three or more years, with falling of the hair, gummatous growths in the viscera, muscles, bones, or skin—so-called *tertiary lesions*.

Etiology: From a bacteriological standpoint the etiology of syphilis is not clear. Micro-organisms have been described by Lustgarten (1884); Eve and Lingard (1886); Disse and Taguchi (1886); Golasz (1894); and Van Niessen (1898). One difficulty encountered by bacteriologists is the fact that animals are not subject to syphilis.

It is known that syphilis may be conveyed by *inoculation*, and that the abrasion need be but slight. Thus infection is transmitted through sexual intercourse, kissing, and through the use of common utensils and vessels for eating and drinking. Physicians have frequently been inoculated in the examination or treatment of cases of syphilis, especially in surgical and obstetrical practice. Occasionally syphilitic infection occurs during circumcision.

Hereditary syphilis may be transmitted from either parent, in whom the disease may be either manifest or latent at the time. Syphilitic infection of a mother at the seventh month of gestation usually does not affect the fœtus, although the fœtus has been reported to be affected as late as the eighth month of gestation. The mother need not necessarily be affected by syphilis transmitted to the offspring from the father, and may afterward nurse the child without becoming

infected, probably through having received a protective inoculation without the development of the disease. The child may convey syphilis to a wet-nurse who has received no such protection. Hereditary syphilis is usually found in the first three months of life, often at the time of birth. Not infrequently abortion or miscarriage is due to inherited syphilis in the fœtus.

A distinction should be made between syphilis acquired with conception and syphilis acquired during intra-uterine life; but for practical purposes hereditary syphilis is usually considered synonymous with congenital syphilis.

Symptomatology: After an *incubation* of two to four weeks the *primary sore*, ulcus durum, appears at the point of inoculation, first as a small red papule, which later breaks down in the centre to form an ulcer. The ulcer has an *indurated base;* hence the term *hard chancre*. The primary sore varies in size, and when small may be overlooked, particularly when located in the urethra. In the female the sore is usually on the inner side of the labia or on the vaginal portion of the cervix. The lymphatics in the neighborhood of the primary sore are enlarged, and suppuration may occur both in the primary sore and in the adjacent lymphatics. The early symptoms of hereditary syphilis are peevishness and irritability at night, harsh and difficult breathing, snuffles, sore mouth, and impaired digestion, with emaciation and the "old man appearance." There may be characteristic eruptions.

Second stage: Usually in from six to twelve weeks constitutional symptoms are observed: *fever*, *anæmia*, *cutaneous and mucous lesions* (macules, papules, and pustules; squamous syphilides, *condylomata*, *falling of the hair*, mucous patches, stomatitis, and *sore throat*), and *affections of the eye* (*iritis*, keratitis, and affections of the optic nerve), sometimes *affections of the ear*, and occasionally epididymitis and parotitis.

Third stage: After a period of several years the so-called *tertiary symptoms* appear. These are chiefly skin-*eruptions*, *gummatous growths* in the viscera, and amyloid *degeneration*.

Other characteristic symptoms of syphilis are the *pains in the bones, especially at night;* the *sunken bridge of the nose*, and the *notched teeth*. The bone-lesions in congenital syphilis

are usually found after the sixth year. Sometimes syphilis involves the kidneys and lungs.

Congenital syphilis may or may not be present at birth. Among the most characteristic symptoms are those due to a *syphilitic rhinitis*, which has given the name "*snuffles*" to the disease. The symptoms of congenital syphilis, when not present at birth, as a rule become *manifest within the first three months*, and resemble those of acquired syphilis, except that the primary sore is not present.

P. Silex recognizes in congenital syphilis three characteristic signs: 1. A choroidea areolaris, in which there are scattered over the fundus of the eye, especially in the neighborhood of the macula, black points and patches, with here and there white spots of various sizes and larger areas with a black border. These represent atrophic colonies in the choroidea, and pigment-patches from the pigment of the stroma and epithelium Vision is impaired from involvement of the retina. 2. A central crescent-shaped excavation in the permanent upper incisors denuded of enamel. 3. Pseudo-scars radiating from the corners of the mouth to the cheek and chin.

Cases of *syphilis hereditaria tarda*, in which the disease was acquired by heredity, but did not become manifest until a long time after birth, have been reported. The existence of such cases is doubted by many, who are inclined to believe that they are really acquired, and that the initial lesion has been overlooked.

Syphilis affects the *bloodvessels* (endarteritis obliterans) and precipitates the changes of age (arterio-sclerosis). In the *liver* syphilis may cause cirrhosis, or the formation of gummata. The *testicle* may be involved in a sarcocele, marked by the absence of pain and fluid, and usually of slow growth.

Diagnosis: A negative history is of little value in diagnosis. Frequently the primary sore is not recognized. A history of an *eruption, falling of the hair, iritis, sore throat*, or *repeated miscarriages or abortions*, may be obtained, and is suggestive. Superficial bone-surfaces (tibia) may be examined for *nodes*. Copper-colored *cicatrices* may be found on the legs, or a scar

may indicate the site of the primary sore. The *testicles* may show atrophy or hardening. Usually *enlargement of the lymphatic glands* may be detected. *Depressed nasal bones* or the presence of *ozæna* may throw light upon a case. The notched teeth of syphilis—the so-called Hutchinson teeth—are due to a disturbance of nutrition, and are not pathognomonic of syphilis, although frequently present in that disease.

Congenital syphilis shows early *snuffles* and a skin-*rash*. About three-fourths of cases develop symptoms of syphilis within the first three months of life; but a negative diagnosis should not be made within less than a year after birth.

Doubtful cases may be cleared up by the *therapeutic test* with mercury and iodides.

Prognosis: Although syphilis is a chronic disease, the prognosis under proper treatment is usually good.

In *hereditary* syphilis the chances are more favorable for the child in cases of infection from the father in which the mother remains healthy.

In 1700 pregnancies destruction of the ovum or fœtus occurred in about one-third of the cases; 1121 children were born alive, and of these children 966 died during the first year of life (Hyde).

Prophylaxis: Segregation would be effective, but is impracticable. Promiscuous sexual intercourse, the habit of kissing, and the use of common drinking-vessels are largely responsible for the propagation of syphilis, and should be prohibited.

Sexual relations should not be permitted until after at least two years' active treatment of syphilis.

Fournier has wisely remarked that nothing is so dangerous to the surroundings as a syphilitic infant (Bulkley).

In cases of hereditary syphilis the father may be treated; the mother must be (Sturgis).

Syphilis—treatment: Some advise excision of the primary sore, since it is a focus of infection. When this is done, medicinal treatment is begun at once; but usually active treatment is deferred until the second stage.

In the treatment of the second stage, mercury, in the form of the ointment, may be given by *inunction*, a drachm a day

for six days, with a bath on the seventh day, the inunction being begun again on the eighth day and continued as before. The patient should be directed to make the applications successively to the forearms, arms, chest, abdomen, thighs, and legs, upon different nights.

Or mercury may be given *internally*, in the form of calomel, or the hydrargyrum cum creta (chalk-mixture, gray powder), with Dover's powder, one grain of each in pills, four to six times a day; or the biniodide of mercury, gr. $\frac{1}{16}$, or the protiodide of mercury, gr. $\frac{1}{5}$, three times a day.

Mercury may also be given by *injection* into the muscles, bichloride, gr. $\frac{1}{3}$ in gtt. xx of water, or calomel, gr. j–ij in ℥xx of glycerin, injected once a week. Mercury may also be given by *fumigation*. *Inhalation* of mercury often gives excellent results; indeed, it is believed that when inunctions of mercury are used the mercury must enter the system through the organs of respiration, since it is well known that very little of the mercury is absorbed through the skin.

During mercurial treatment, *salivation* should be guarded against by keeping the teeth and mouth clean, avoiding acids, green vegetables, and fruit. Should symptoms of salivation supervene, manifested by tenderness of the gums, the use of mercury may be suspended, or potassium chlorate may be given, a teaspoonful of the saturated solution every two hours.

Congenital syphilis may be treated with mercury by inunction or internally, in the form of the hydrargyrum cum creta. Later manifestations may call for the "mixed treatment," Gilbert's syrup (biniodide of mercury, gr. j; iodide of potassium, half an ounce; water, two ounces), gtt. v–x three times a day, gradually increased to tolerance.

Syphilis in infants may be treated indirectly through the administration of mercury and the iodides to the nurse.

Children with hereditary syphilis almost invariably die if taken from the breast. This has generally been attributed to decreased nutrition, but possibly may be due to some substance of therapeutic value in the mother's milk.

In the treatment of the *third stage* the iodides, especially the iodide of potassium, takes the place of the mercury used

in the second stage. The patient may begin with gr. x, gradually increased to gr. xxx or more, largely diluted, in milk or water three times a day.

In all cases it is advised to *continue treatment at least two years.* Some believe that this length of time may be made shorter by the use of injections of mercury.

CHANCROID (Soft Chancre; Ulcus Molle).

A venereal sore, that appears within a day after infection as a red spot upon the glans penis. At first a papule, it becomes a day or two later a vesicle. Rupture of the vesicle forms an ulcer, characterized by a profusely suppurating base. The ulcer causes infection of contiguous structures, and often spreads through the lymphatics to cause suppuration of the lymphatic glands—bubo. Inoculation-experiments are successful in apes and man.

Diagnosis: The rapid onset, the absence of induration of the base of the ulcer, and the fact that there is no general infection, differentiate soft chancre (chancroid) from the hard chancre (true syphilis), and make recognition of the disease easy.

Treatment: When seen early the ulcer should be destroyed with the cautery or strong caustics, caustic potash or zinc chloride, or with fuming nitric acid. Later, mild treatment is best. The ulcer may be cleaned and covered with iodoform, europhen, dermatol, calomel, or bismuth.

GONORRHŒA (Blennorrhœa; Urethritis Specifica; Clap; Tripper (German)).

Definition: Infection of the urethra by the gonococcus.

Etiology: The specific cause is the gonococcus, micrococcus gonorrhœæ, a diplococcus discovered by Neisser (1879). Infection occurs usually through impure intercourse, to cause greater or less involvement of the genito-urinary system. Thus there may be produced a specific urethritis, affecting sometimes the posterior urethra; prostatitis, adenitis (bubo), orchitis; salpingitis, oöphoritis, metritis, peritonitis; cystitis,

ureteritis, pyelitis, and nephritis. Careless manipulations may permit inoculation of the anus, condylomata; or of the eye, conjunctivitis. During parturition from an infected mother a child may be inoculated, to cause most frequently affection of the eye, conjunctivitis, *blennorrhœa neonatorum;* less frequently, vaginitis or stomatitis.

The gonococcus has been found in the blood and upon the valves in cases of gonorrhœal endocarditis. Pericarditis, pleurisy, and myocarditis are rare. Gonorrhœal arthritis is more frequent. Sometimes pure cultures of the gonococcus may be obtained from infected joints. The knee-joint is the more frequently involved. Often the gonococcus opens the way for secondary invasion by other micro-organisms (see Septicæmia).

Gonorrhœa—symptomatology: *Incubation*, two or three days. The symptoms come on with *dysuria*, painful erections (*chordee*), a *muco-purulent discharge*, becoming later a more or less continuous discharge of pus—*pyuria*. In bad cases there may be a bloody discharge, due to the destruction of tissue. As a rule there is *fever*, probably caused by the absorption of toxins. *Infection of the lymphatics* may cause enlargement of the glands in the groin. Sometimes there is *affection of the testicle*, especially of the epididymis, with effusion into the tunica vaginalis. *Condylomata* may appear upon the glans penis or perineum. Infection of the glands of Cowper is announced by a sense of weight and pain, and the appearance of a tumor, in the median line of the perineum. Suppuration with discharge into the urethra may lead to the formation of fistulæ.

Among the *nervous phenomena* are insomnia, headache, priapism, and emotional disturbances; the individual becomes irritable, sometimes dejected.

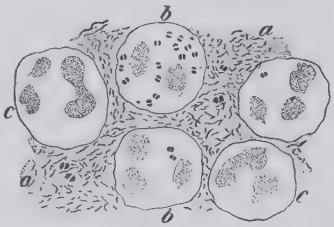
The chief **complications and sequelæ** are prostatitis, adenitis, peri-urethral and prostatic abscess, orchitis; vulvitis, vaginitis (leucorrhœa), metritis, salpingitis, oöphoritis, sometimes peritonitis; cystitis, ureteritis, pyelitis, nephritis; arthritis, endocarditis, septicæmia, conjunctivitis, iritis; pericarditis, pleurisy; synovitis, and stricture.

Diagnosis: The symptoms, especially dysuria and pyuria,

may arouse suspicion; but a positive diagnosis may be made only upon disclosure of the gonococcus (Fig. 7).

Examination for the gonococcus: The gonococcus is a "biscuit-shaped" or kidney-shaped *diplococcus*, arranged with its concavities in apposition, separated by a narrow zone. The organism is found *within pus-cells* and upon epithelial cells, and is *decolorized by Gram's method.* Cultivation is difficult,

FIG. 7.



Gonococci in the secretion from the urethra in recent gonorrhœa. Cover-glass preparation stained with methylene-blue. *a*, mucus with separate cocci and diplococci; *b*, pus-cells with diplococci; *c*, pus-cells without diplococci (Ziegler).

but may be accomplished upon human blood-serum, or upon the ordinary nutrient agar to which urine has been added.

The gonococcus shows an affinity for the basic aniline dyes, especially methyl-violet, gentian-violet, and fuchsin. Probably methylene-blue is the best stain when searching for the organism in pus. Beautiful double staining may be done with methylene blue and eosin, or with Ziehl's solution of fuchsin and methylene-green.

Prognosis: Good, under proper and persistent treatment. The occurrence of complications makes the outlook less favorable.

Gonorrhœa—treatment: The bowels should be kept open. A light diet is best. Fluids, but not alcohol, should be taken in abundance. Rest in the recumbent posture is advisable. The testicles should be supported in a light bandage. Priapism calls for sponging of the organ with cold water and the internal use of the bromides, camphor, lupulin. Dysuria may be relieved by salol, the salicylates, phenacetin, or in severe cases by suppositories of opium or belladonna.

After subsidence of the acute symptoms oil of copaiba

or sandalwood may be given internally. Later the urethra may be treated locally with mild solutions of the acetate of lead or zinc, nitrate of silver, protargol, chloride of zinc, or alumnol.

Posterior urethritis calls for the deep prostatic injection of nitrate of silver with an Ultzman catheter; or iodoform may be injected, a 10 per cent. solution in pure glycerin. The odor may be disguised with vanillin or coumarin, 1:10. The remedy is injected, a syringeful at a time, immediately after the patient has voided the urine. Complications may need special treatment. Cystitis is best met by washing out the bladder with mild solutions of boric acid, permanganate of potassium, nitrate of silver, or protargol.

Obstinate cases may sometimes be cleared up by the use of the endoscope, whereby a localized inflammation may be discovered and treated directly by local applications.

Buboes may be treated by the injection in two places of a 1 per cent. solution of the benzoate of mercury. This treatment will sometimes prove successful when incision has failed, and has been reported to succeed even after suppuration has begun.

GLANDERS (Farcy; Rotz (German); Morve (French)).

Definition: An infectious disease, acute or chronic, communicated to man from the domesticated animals, especially the horse, and characterized by nodules, particularly in the nose and beneath the skin. The disease does not affect cattle nor swine.

Etiology: Glanders is caused by the *bacillus mallei*, discovered by Löffler and Schütz (1882). The disease occurs in man chiefly through inoculation from diseased animals; occasionally from man. The infection is disseminated through the lymphatics.

Glanders—symptomatology: *Incubation*, usually three or four days. The *point of inoculation shows swelling and redness* with *inflammation of the lymphatics*. *Nodules* form *in the nasal mucous membrane* and break down to form *ulcers*, from which there is a muco-purulent *discharge*. *Papules*, which

soon become *pustules*, appear on the face and over joints. The patient experiences *chilly sensations*, *fever*, *headache*, and *prostration*.

Chronic glanders present nasal ulcers, and often also laryngeal symptoms. The disease may be mistaken for chronic coryza.

Farcy, in animals, presents a phlegmonous inflammation of the skin at the point of inoculation. With inflammation of the lymphatics there are formed enlargements along their course, to constitute the *farcy "buds."* These usually soon show suppuration. As a rule this form of the disease reaches a fatal termination in about two weeks. In the *chronic form* farcy shows localized tumors, which break down, sometimes forming deep ulcers. There is not much involvement of the lymphatics. The duration of this form of the disease may be for months or years.

Glanders—diagnosis: The *occupation* of the patient—possibility of contact with diseased animals—may lead the physician to suspect the disease in the presence of a nodular eruption or ozæna.

Mallein, a product of the glanders bacillus, is used in the diagnosis of glanders, much as tuberculin is used in the diagnosis of tuberculosis. This is of especial value when the disease is located in some recess of the body, as in the lungs, where it may not be brought under direct observation. Should mallein not be accessible, *a male guinea-pig may be inoculated*, or better several of them. The inoculation is made into the abdominal cavity. Two to five days after inoculation the testicles and their sheaths become swollen and purulent.

Prognosis: Cases of acute glanders usually terminate fatally in about eight or ten days. Chronic glanders may last for months, and sometimes results in recovery.

Glanders—prophylaxis: Diseased animals should be killed, and as far as possible individuals should be protected from the danger of inoculation. To this end, after death cremation of the bodies of both men and animals is advisable in all cases.

Treatment: As far as possible, especially in early cases, the

diseased tissue should be removed by the knife or cautery, and the parts treated antiseptically.

The value of mallein as a therapeutic agent has not been definitely determined.

FOOT-AND-MOUTH DISEASE.

Definition: A disease, involving especially the mouth and extremities, sometimes the udder and teats, that occurs most frequently in cattle, sometimes in other animals, and is occasionally *communicated to man* through the ingestion of *milk* and other dairy-products from diseased animals, or directly by *inoculation*.

Some believe that *aphtha* is an expression of this disease in man.

Foot-and-mouth disease—symptomatology: *Incubation*, two to ten days. The chief *prodromata* are pains in the head and limbs; *fever*, 100° to 103° F.; *malaise;* vertigo; and frequently a sensation of formication in the hands and feet. An *eruption* then appears *upon the mucous membrane of the mouth and nose*, consisting of vesicles with at first clear contents, which later become turbid. The vesicles may burst and crusts form. There is swelling of the tongue, lips, nose, and eyelids. Often vesicles appear upon the *fingers*. An eruption of vesicles, discrete or confluent, may appear upon various parts of the body or become general. With appearance of the eruption the temperature usually falls. The affection of the mouth causes *difficult deglutition and speech*, sometimes difficult respiration.

The disease may last from five to eight days in mild cases; as long as eight weeks in severe cases.

The principal *complications* are diarrhœa; hemorrhages from the mouth, bowels, or kidneys; sometimes bronchitis and catarrhal pneumonia; occasionally spasms and paralyses.

Foot-and-mouth disease—diagnosis: The diagnosis rests on the *evidence of direct or indirect transmission* of the disease from infected animals, *in conjunction with the symptoms* of the disease. *A positive diagnosis may be made by inoculating an animal*, best a goat, with the contents of the vesicles from a

suspected case. As the name indicates, the disease is peculiar in that the *eruption usually appears only upon the mouth and extremities.*

The *differential diagnosis* concerns chiefly other forms of stomatitis, scurvy, measles, typhoid fever, septicæmia, and rarely syphilis.

Prognosis: Usually good. Death may occur in delicate children, the aged, or individuals weakened by disease or other cause.

Foot-and-mouth disease—prophylaxis: Diseased animals should be isolated. Individuals who have abrasions of the skin should not come in contact with diseased animals or individuals. The dairy-products and meat from infected animals should not be used, or should at least be subjected to sterilization by heat before being used.

Treatment: Largely symptomatic. Siegel uses salicylate of sodium internally. Among the many remedies used *locally* are chlorate of potassium, borax, alum, lead, nitrate of silver, salicylic acid or zinc paste, dermol, creolin, lysol, and permanganate of potassium. The patient should be kept comfortable by the use of opium, best in the form of Dover's powder, chloral, the salicylates, and phenacetin.

TYPHOID FEVER (Typhus Abdominalis; Enteric Fever; Nervenfieber (German)).

Definition: An acute infectious disease, due to a special bacillus, characterized by hyperplasia and ulceration of the intestinal lymph-glands (Peyer's patches), and enlargement of the mesenteric glands and of the spleen. Nervous symptoms are marked. There are headache and hebetude, a cloud about the brain, constituting the *status typhosus.* The disease shows more or less characteristic fever, eruption, stools, and meteorism. Sanarelli defines typhoid fever as an infection of the lymphatic system by the typhoid bacillus.

History: First recognized as a separate disease by Pierre Bretonneau (1813); named "Typhoide" by Louis (1829); separated from typhus fever by Gerhard, of Philadelphia (1837). The *bacillus typhosus abdominalis* was described by Eberth, and observed and photographed by Koch (1880).

Etiology: The *bacillus typhosus abdominalis* is generally recognized as the specific cause of typhoid fever.

Typhoid fever seems to show *preference for the temperate climate* and prevails especially in the *fall months*. Hot, dry weather seems conducive to a dissemination of the disease, probably through greater contamination of the water-supply. The bacillus of typhoid fever gains entrance to the body chiefly through the water-supply and milk.

Typhoid bacilli, when mixed with fat, oil, or butter, are not killed by fresh gastric juice, and thus may pass into the intestine.

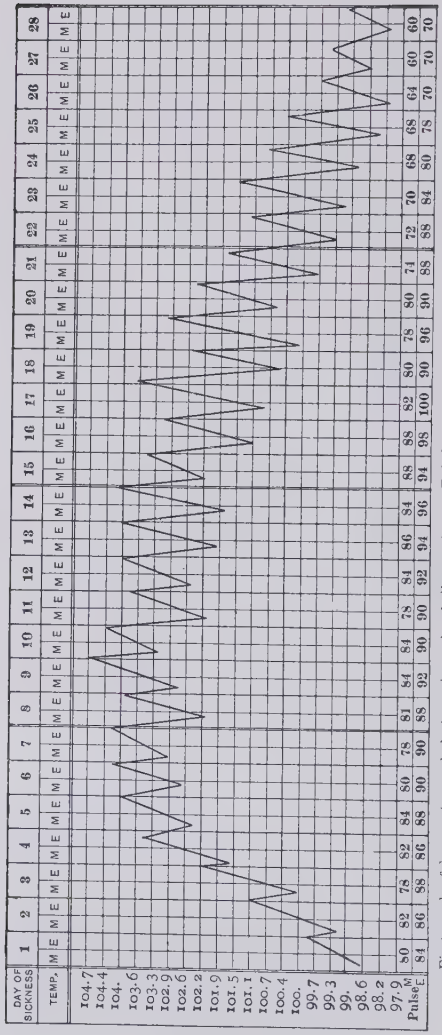
The disease occurs especially in *youth* and *adolescence;* rarely in infancy and age.

Typhoid fever—symptomatology: *Incubation*, four to twenty-three days; usually *about two weeks*. Often during incubation there are *lassitude, early fatigue*, and especially *lack of concentration*.

The onset of typhoid fever is insidious. Among the *prodromal symptoms* are *languor, headache, coated tongue, anorexia, nausea, epistaxis, pain in the back and legs*, sometimes in the abdomen; *chilly sensations*, rarely rigor, and sometimes vertigo. These symptoms continue to increase until the patient is forced to his bed, which is usually reckoned as the *first day* of the disease.

Often during the *first week* of the disease the *temperature* (Fig. 8) *shows the so-called step-ladder rise*, being a degree or more higher in the evening than the previous evening, and a degree higher in the morning than the previous morning, reaching by the fifth to the seventh day 103° to 104° F. The *pulse* is quickened, 100 to 110, of full volume but low tension, sometimes becoming dicrotic. With the high fever there may be *delirium*. By the latter part of the first week the *spleen is noticeably enlarged* and the *rose-colored lenticular spots* are first seen on the skin, as a rule, in the region of the diaphragm. The *tongue is coated* white, with clean, bright-red margins and tip. There are usually *meteorism and diarrhœa, sometimes constipation*. After the discharge of the normal contents of the intestine the *stools* assume the "pea-soup, ochre-colored" appearance, sometimes colored with

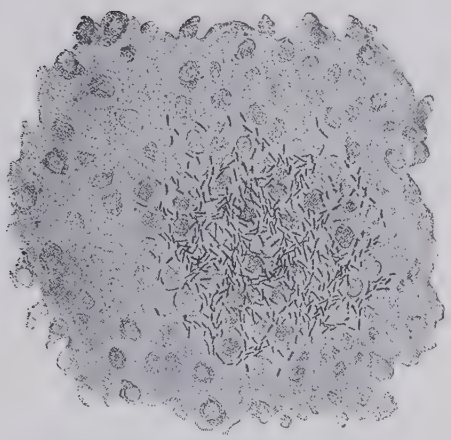
FIG. 8.



Temperature in unmodified typhoid fever.

blood. Blood may be found with the microscope in almost all cases. The *urine* is diminished in quantity, the urea

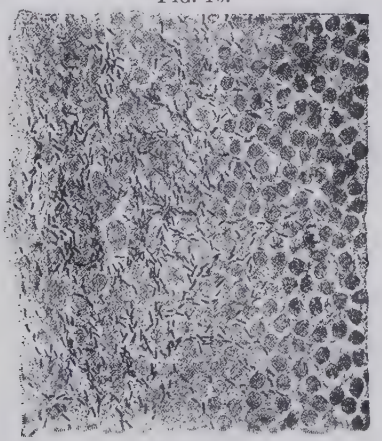
FIG. 9.



Typhoid bacilli from a section of the human spleen, tenth day of enteric fever (Charcot).

increased, chlorides diminished, and frequently there is a trace of albumin. There may be a slight cough, a symptom of *bronchitis*, very early in the course of the disease.

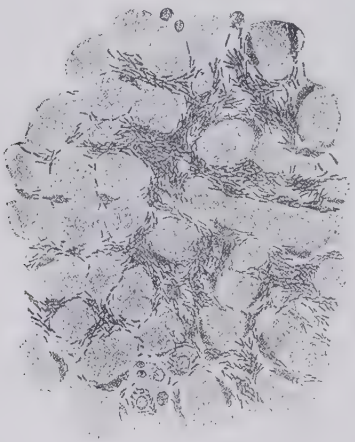
FIG. 10.



Typhoid bacilli in a Peyer's patch before ulceration (Charcot).

During the *second week* the *fever* continues on a high plane with slight morning remissions. The other symptoms become more pronounced. The *pulse* varies, 90–120, and is less dicrotic. Headache gives way to *mental torpor* and *dulness*.

FIG. 11.



Human liver, tenth day of enteric fever (Charcot).

The *tongue* is dry and covered with sordes. Toward the end of this week there is danger of *perforation and hemorrhage*.

In *mild cases* the symptoms may begin to improve by the end of the second week.

As a rule the symptoms continue during the *third week* much the same as during the second week, only more severe. There may be low muttering delirium, stupor, coma-vigil, and picking at the bedclothes in bad cases. The emaciation and *loss of strength* are more marked. The *fever* shows greater morning remissions, a beginning lysis. *Pulse*, 110–130. During this week *perforation and hemorrhage*, *bedsores*, *pneumonia*, and *heart-failure* are the complications to be most feared.

Usually during the *fourth week* the *temperature* reaches the normal by a gradual descent—lysis—and all the other *symp-*

toms show improvement. In *severe cases* the picture presented during the third week may be continued into the fourth or even the fifth week, only becoming worse through the weakness of the patient. As a rule a marked improvement in the patient's condition occurs during the fourth week and convalescence begins. With the beginning of convalescence the patient shows a considerable *increase in appetite.*

The more important **complications** of typhoid fever are *perforation, peritonitis, hemorrhage,* and parenchymatous degenerations of muscles. Thus the heart-muscle may be affected so as to cause *heart-failure;* or the diaphragm may be rendered incompetent. Many, if not most, cases are affected secondarily by *septicæmia.*

Typhoid fever—diagnosis: In cases that come under observation early the *anorexia, headache, weakness, epistaxis, diarrhœa, gradual rise of temperature,* and *roseola,* and later *tympanites* and *enlargement of the spleen,* are usually sufficient for diagnosis in typical cases.

The blood-test for typhoid fever (Widal test) is almost, if not absolutely, pathognomonic of infection by the typhoid bacillus. Unfortunately, the reaction is found sometimes for a long time after recovery from typhoid fever. Apple and Thornbury report a case in which the reaction persisted thirty-one years. But usually the reaction disappears within a year. The history should not be implicitly relied upon in excluding a previous attack, since typhoid fever may have been mistaken for some other disease. Further, in a few instances infection by the typhoid bacillus has occurred in other parts of the body than the alimentary tract. But as a rule the history is clear, and the blood-test remains the best single sign we possess.

Method: The best and simplest way to make the test (Widal) is with dried blood. A drop of blood is collected, from the finger-tip or lobe of the ear, upon a piece of glass and permitted to dry. When the examination is to be made a particle of the dried blood is added to just sufficient water to cause indistinct coloring. Of this an ordinary platinum loopful is added to a similar quantity of an emulsion of the typhoid bacillus in a hanging drop under the microscope.

The culture of the typhoid bacillus should be twelve to twenty-four hours old, made from a stock culture a month old. The emulsion of the typhoid bacillus is made by adding to a drop of normal salt solution (0.6 per cent.) a trace of the culture of the typhoid bacillus.

Reaction: When diluted typhoid blood is added to an emulsion of the typhoid bacillus, the bacilli are observed under the microscope to become agglutinated together in little clumps, and to lose their motility. To be positive, the reaction should be present within fifteen minutes.

The **diazo-reaction** of Ehrlich does not furnish so reliable evidence as the blood-test, but is more readily made and often sheds a valuable side light upon a doubtful case. The *reagents* necessary are (1) a 0.5 per cent. solution of sodium nitrite; (2) sulfanilic acid solution, composed of a 5 per cent. solution of hydrochloric acid in distilled water and sulfanilic acid to saturation; (3) ammonia.

Method: To 3 c.c. of urine add one drop of the sodium nitrite solution. Shake. Add 3 c.c. of the sulfanilic acid solution. Shake. Add an excess of ammonia.

The *reaction*, when positive, is marked by a rose-red to a dark-red color, which persists also in the foam. A brownish-yellow color is negative.

In typhoid fever the diazo-reaction is present from the middle of the first week up to the ninth day and longer, but not after the third week. Absence of the reaction before the ninth day excludes typhoid fever, at least in an average or grave form.

The **typhoid bacillus** may be isolated from the *urine*, *feces*, or *blood*, especially blood withdrawn from the *spleen*, rarely from the rose-colored spots. But the withdrawal of blood from the enlarged and friable spleen, in which the bacilli may most frequently be found, is not without danger, through rupture of the spleen and hemorrhage.

During the first week typhoid fever should be **differentiated** especially from febricula, influenza, and the exanthematous diseases common among children. When a case first comes under observation late in the course of the disease the differential diagnosis concerns especially malaria, acute miliary

tuberculosis, appendicitis, peritonitis, trichinosis, and, especially in children, entero-colitis.

Typhoid fever—prognosis: The treatment with cold baths has reduced the mortality from about 30 per cent. to about 5 per cent. The mortality is especially high in the intemperate, gouty, and corpulent. Recovery is the rule, with very few if any exceptions, in cases that are seen early and treated faithfully with the cold bath whenever the temperature reaches 103° F. in the rectum. The prognosis will depend upon the height and duration of the fever, the presence of complications, the time the patient comes under treatment, especially the time the patient takes to his bed, and the degree of toxæmia, as manifested by the strength of the heart and the presence of nervous symptoms.

Prophylaxis: The ingesta, especially the milk and water, should be clean—*i. e.*, not contaminated by the excreta of typhoid-fever patients. Or if such food and drink must be used, it should first be subjected to the temperature of boiling water.

The excreta (stools and urine) of the patient should be disinfected, best by fire, or carbolic acid (1 : 20), or bichloride of mercury (1 : 1000). Clothing contaminated by the discharges should be sterilized.

Typhoid fever—treatment: Proper nursing is of the greatest value. The patient must remain in bed, and under no circumstances arise from the recumbent posture until the temperature has remained normal at least three successive days. This implies the regular use of the bed-pan and urinal. The food should be fluid, chiefly milk, which may be given raw or boiled, hot or cold, sometimes coagulated with rennet or in the form of koumyss, kephir, or matzoon; occasionally buttermilk or wine-whey, milk-punch, or egg-nog. The milk-diet may be varied with clear soups and broths, made from beef, mutton, veal, or chicken; or consomme, with or without vegetables, rice, or barley, carefully strained. The patient may receive also oyster-soup, clam-juice, strained barley-gruel, and meat-juice. At any rate, the diet must be liquid. To guard against continued infection, it may be necessary to boil the water and milk or to secure these articles from a different source.

The patient should be placed under good *hygienic* surroundings, with plenty of fresh air and sunshine, and secluded from society. During convalescence the visits of friends may at first be limited both in number and duration. Throughout the illness small quantities of food and drink should be offered at definite intervals, usually every two or three hours.

Of *drinks* pure water is best, sometimes in the form of iced tea, lemonade, or barley-water. At times the juice of an orange or lemon is very grateful.

Various "specifics" have been recommended from time to time; among them guaiacol, calomel, bichloride of mercury, carbolic acid, sulphuric acid, iodine, chlorine, quinine, salol, the salicylate of bismuth, boric acid, turpentine, oil of eucalyptus, thymol, camphor, and beta-naphthol, but none of these has been generally accepted.

Early in the course of typhoid fever it is best to administer *calomel* or castor-oil to empty the intestinal tract, especially when there is constipation.

In the way of *specific medication*, the transfusion of blood from convalescents has been practised by Hammerschlag; injections of blood-serum from convalescents by Hughes and Carter; the serum of animals rendered immune through inoculation by Beumer and Peiper (sheep); and Klemperer and Levy (dogs), with results sufficiently satisfactory to call for further experimentation along this line.

Fränkel and Manchot, in fifty-seven cases, obtained promising results by injection of sterilized thymus bouillon-cultures of the typhoid bacillus deep into the muscles of the back. The cases in which the injections were continued showed an amelioration of the constitutional symptoms, with an early fall of temperature, increase in the quantity of urine, and a cessation of diarrhœa. Rumpf reported somewhat similar results in the treatment of thirty cases of typhoid fever with similar cultures of the bacillus pyocyaneus.

Loss of appetite and strength may be met probably best with the tincture of nux vomica before meals. In the presence of fever the gastric juice is not formed so readily, and it is best to give dilute hydrochloric acid after meals.

Slight fever may be let alone; higher fever, above 103° F.

in the rectum, calls for the *cold bath*, which lowers the temperature and strengthens the heart. The bath should be at a temperature of 68° F.; or may be begun at a higher temperature and gradually lowered. The duration of the bath (five to twenty minutes) must be sufficient to lower the temperature of the patient two degrees. The bath may be repeated every two hours should the temperature reach 103° F. in the rectum.

Cold sponging, the application of cold compresses or of ice, the cold pack, etc., are poor substitutes for the cold bath; but are often useful. Beneficial results may sometimes be obtained by the judicious employment of antipyretic drugs, of which lactophenin is probably the safest.

Tympanites and abdominal pain may be relieved by turpentine stupes. For meteorism, in the presence of a dry tongue, sordes, and muttering delirium, turpentine may be given internally or by enema.

Excessive diarrhœa—more than three or four stools a day—may be controlled by enemata of starch and opium, or the administration per os of bismuth and Dover's powder, or a combination of tincture of opium, hydrochloric acid, and camphor-water. Constipation may be relieved by enemata repeated every three or four days if necessary.

Hemorrhage calls for absolute rest, restricted diet, ice both internally and externally, the administration of opium and acetate of lead. Collapse may be met by the injection of the physiological salt solution, 0.6 per cent., into a blood-vessel, the rectum, or the subcutaneous tissue.

For *peritonitis* morphine may be given hypodermatically. *Perforation* may demand laparotomy, which has saved three cases out of seventeen reported.

Weakness of the heart calls for stimulation with alcohol or digitalis internally; camphor, strychnine, or ether hypodermatically, to bridge an impending collapse.

During convalescence, after the temperature has been normal ten days, the patient may gradually return to a solid diet.

TYPHUS FEVER (Typhus Exanthematicus).

Definition: An acute infectious disease, probably due to a specific micro-organism, characterized by sudden onset, a

peculiar eruption, which is usually present, and, as a rule, termination about the fourteenth day by crisis.

History: "Typhus fevers" were recognized by the older clinicians; but they did not separate typhus fever from a number of other fevers, notably typhoid fever and relapsing fever. Typhus was differentiated from typhoid fever by Gerald and Pennock, of Philadelphia (1836), and the non-identity of the diseases confirmed by Jenner, of London (1849–51).

Etiology: Various micro-organisms have been found in typhus fever, but none of them has been proven to be the cause of the disease. Balfour and Porter found a diplococcus in several cases post-mortem; and in fifteen out of nineteen cases of typhus fever examined during life, in which the diplococcus was the only organism present.

Among the predisposing causes are overcrowding, bad ventilation, poverty, famine and scarcity of food, and intemperance.

Typhus fever—symptomatology: The period of *incubation*, variously given at from a few hours (Huss) to as long as thirty-one days (Hutchinson), is usually about twelve days.

The *invasion is short*, one to three days, *and abrupt*, beginning with *chilly sensations*, sometimes with a distinct *chill*. There are *malaise*, later great *prostration*, headache, vertigo, anorexia, general soreness of the body, and pains in the loins and extremities, especially the lower extremities. The *tongue*, large and pale, presents at first a white coat, which later becomes darker in color. The *face* is flushed and dusky; the conjunctivæ show a well-marked uniform congestion. Usually the hands show *tremor*. The *urine* is small in quantity and high-colored, specific gravity possibly 1030. The *temperature* reaches 102°–105° F. within a day. The pulse is rapid and compressible. Usually the *abdomen* presents nothing abnormal. *Constipation* is the rule. Sometimes there is nausea, more rarely vomiting.

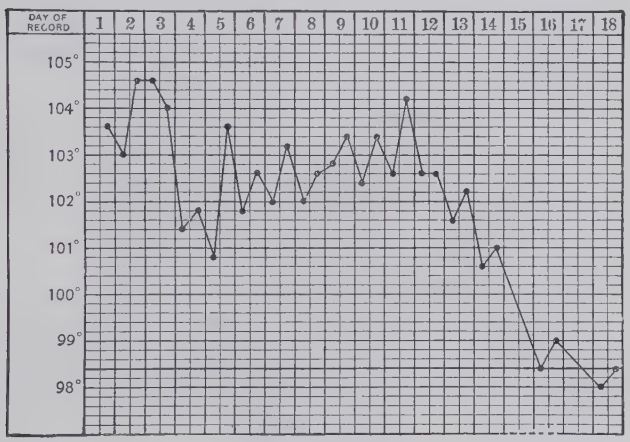
The *eruption* appears on the third, sometimes as late as the sixth day, *first as a dark punctate measly rash, which disappears on pressure, to reappear when the pressure is removed*. The eruption is found *first upon the abdomen*, later on the

arms and thighs, more rarely on other parts of the body, the face, and neck. An eruption under the skin, in addition to the rash just referred to, constitutes the *mulberry rash of Jenner.*

Later, about the fifth day, the measly rash becomes darker in color and does not disappear on pressure, due to capillary hemorrhage and the deposit of pigment. About the tenth day true *petechiæ* appear, which do not disappear after death. The eruption usually disappears in eight or ten days.

The *temperature* rises rapidly during the first week, often reaching 103° F. or more in a day or two, and remains high,

FIG. 12.



Typhus fever.

sometimes with gradual descent, until about the end of the second week, when the temperature drops suddenly, by *crisis*, to normal or subnormal. In uncomplicated cases a *remission* occurs early in the second week. As in typhoid fever, the temperature in typhus fever shows a slight diurnal variation.

Diagnosis: The knowledge of the existence of the disease in the neighborhood is of value. Isolated cases may present great difficulties in diagnosis, especially in the absence of eruption. The *sudden onset, great prostration*, with the *dense*

cloud about the brain, and *peculiar eruption*, appearing about the third day and sparing the face, and the *crisis at the end of the second week*, are characteristic.

Typhus fever should be *differentiated* especially from typhoid fever, cerebro-spinal meningitis, pneumonia, small-pox, uræmia, and severe cases of yellow fever.

Prognosis: Children rarely die of typhus fever; the mortality among the aged and intemperate is high. Different epidemics have given a mortality of from 12 to 20 per cent. Much depends upon the strength of the heart and the degree of toxæmia.

Prophylaxis: There should be early isolation of typhus fever patients, and later thorough disinfection, with fire, formaldehyd, bichloride of mercury, or sulphur, of the room and of all articles with which the patient came in contact.

Treatment: The treatment of typhus fever is much the same as of typhoid fever. Special stress should be laid on hydrotherapy, especially cold baths. The heart must be supported, best with alcohol, digitalis, and nitroglycerin.

RELAPSING FEVER (Recurrent Fever).

Definition: An acute infectious disease, caused by the *spirochæta Obermeieri*, characterized by an intermission at the end of the first week, with the disappearance of the fever and symptoms, followed by one or more relapses.

Etiology: The specific infectious agent of relapsing fever is the spirochæte (spirillum) discovered by Obermeier, in the blood during fever, in 1873. Relapsing fever is contagious directly and possibly indirectly through fomites. The predisposing causes are similar to those in typhus fever.

Relapsing fever—symptomatology: *Incubation* one to fourteen days, *usually five to seven days*. For a day or two there may be anorexia, lassitude, headache, and vertigo.

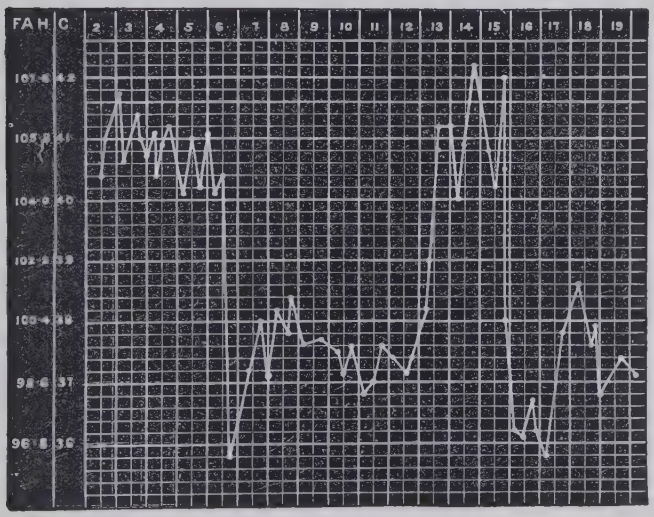
Invasion as a rule is abrupt, with a *chill* followed by a rise of temperature, frequently 104° F., which in a day or two reaches 105°–106°. The pulse becomes rapid, 110–130, full and strong. The *spleen shows early enlargement*. There are giddiness, headache, and severe *pain* in the muscles of the

trunk and extremities, especially in the calves of the legs. Some cases begin with nausea and vomiting.

The face is flushed, the tongue coated white. The patient complains of thirst. Often there is *jaundice*. Some cases show an eruption of petechiæ. The urine is scanty, dark colored, albuminous, with high specific gravity, containing bile when there is jaundice, sometimes blood.

With the *crisis* the temperature falls suddenly to normal or subnormal. The pulse drops to 70 or less. Often just before

FIG. 13.



Relapsing fever (Murchison).

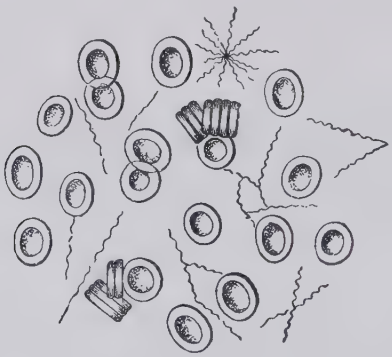
the crisis the temperature becomes higher and there are sweating, diarrhœa, and epistaxis, sometimes an appearance of the menstrual flow, sometimes delirium.

Rarely does convalescence proceed uninterruptedly. As a rule about a week, four to fourteen days, after the crisis there is a *relapse*, resembling the original attack, but somewhat shorter in duration, the *second crisis* appearing usually in

three to five days. Recovery may now take place, or there may be as many as three or even four relapses.

Diagnosis: The diagnosis of relapsing fever rests upon the discovery of the *spirochæta Obermeieri*, which is found upon microscopic examination of the fresh blood during the fever.

FIG. 14.



Spirochætes of relapsing fever in the blood.

Differential diagnosis, especially during the onset of the disease, concerns typhus fever, yellow fever, and smallpox. Cases seen later may be confounded with typhoid fever.

The *intermissions and relapses* are characteristic.

Prognosis: Death rarely occurs, except through collapse or complications, especially pneumonia.

Treatment: Löwenthal treated 131 cases of relapsing fever with *antispirochætic serum*. Thirty-four of the cases were thoroughly treated, with but one death. Compared with 152 cases not subjected to specific treatment, the use of the serum lowered the mortality about one-half, markedly lessened the number of relapses, and shortened the duration of the disease.

Symptomatic treatment: Pain may call for opium; a threatened collapse for stimulants—alcohol, camphor, ether, strychnine, ammonia, digitalis.

YELLOW FEVER.

Definition: An acute infectious disease, caused by a specific micro-organism, characterized by icterus and hemorrhage from the mucous membranes.

Etiology: The specific infectious agent is the *bacillus icteroides* (Sanarelli), which resembles closely and is possibly identical with the micro-organism described by Sternberg as the bacillus X.

The *bacillus icteroides* is a slender bacillus, 2–4 μ long, motile, ciliated, facultative, anaërobic. The bacillus is decolorized by Gram's method, grows in the usual culture-media, causes fermentation when grown upon a culture-soil containing sugar, and is pathogenic, producing in man, monkeys, and dogs the symptoms of yellow fever. In many cases the bacillus is not found, probably because it is overrun by secondary infection, especially by the pyogenic streptococci and staphylococci. The bacillus seems to thrive better and show greater virulence in the presence of a certain fungus, an aspergillus. This fungus thrives only in warm weather, which may explain the prevalence of yellow fever during the warm season (Lacerda).

The bacilli occur in small numbers, but produce a powerful toxin, the *amaril poison* of Sanarelli.

Yellow fever *prevails* especially along the sea-coasts and prefers unsanitary conditions and a hot climate. The bacillus icteroides resists well both drying and the action of sea water, and its growth seems to be favored by the moulds. One attack of yellow fever usually confers immunity.

Yellow fever—symptomatology: *Incubation* lasts from a few hours to five days. Preceding the attack there may be some malaise, anorexia, lassitude, headache, vertigo, and indisposition, both physical and mental; or all these symptoms may be entirely absent. As a rule the attack begins suddenly with a *chill* or chilly sensations. Sometimes the onset is insidious. There are *fever* and more or less *pain in the head, especially in the frontal and supraorbital regions.* The eyeballs are painful, and there may be photophobia. The patient complains of *pains in the loins and calves of the legs.* After the chill the

temperature rises rapidly. The *face is flushed*, often congested and swollen. The *conjunctivæ are injected*. The skin is dry and hot, and there may be considerable restlessness and jactitation.

Typical cases usually present *three stages*. The first stage begins with the initial paroxysm and lasts two to five days. This stage includes the *high temperature*, which is usually highest on the first day. The second stage, beginning after the fall in temperature, shows often *subnormal temperature* and great *prostration*, lasting a few hours or days. The third stage is the period of *convalescence*, during the early part of which there is a *remittent fever*.

Jaundice, which is not present in all cases, begins toward the end of the first stage, is most intense during the second stage, and may last far into convalescence. Sometimes jaundice does not begin until the second or third stage, when it usually lasts longer. The *urine*, scanty and high-colored, early shows *albumin*. There may be *uræmia*. There are gastric distress, vomiting, and hæmatemesis (*black vomit*), due to toxæmia. Sometimes the "black vomit" is absent throughout the course of the disease, even in severe cases.

Yellow fever—diagnosis: In the blood-test, with the bacillus icteroides, the reaction of paralysis and agglutination is present as early as the second day of the disease. *Albuminuria* is usually present by the third or fourth day, and with the *temperature* and *pulse* is of value in diagnosis. Later the appearance of *icterus*, without enlargement of the spleen, helps in differentiation. The *history* of the patient, especially regarding the place of *residence* and *absence of previous attack*, and the knowledge of the *presence of an epidemic*, may aid in the individual case.

The **prognosis** of yellow fever is more favorable among the natives of regions where the disease exists continuously than among those not "acclimatized," and is better for women and children than for men. The prognosis may said to be favorable when the temperature does not rise above 103° F. by the end of the second day, and assumes gravity in proportion to the height of the fever at this time. The mortality varies in

different epidemics. The majority of deaths occur during the first week of the disease.

Yellow fever—prophylaxis: Patients should be isolated. The excreta, and all articles coming in contact with the patient, should be thoroughly disinfected, best by fire. Susceptible individuals, as far as practicable, should avoid infected regions.

Yellow fever—treatment: Fitzpatrick (1899), working at the instance of Doty, injected horses with the filtrate of cultures of the bacillus icteroides. The blood-serum of the horses was then found to prevent the lethal effects in guinea-pigs of inoculations with the bacillus icteroides, that proved fatal in control-animals.

Early in the course of the disease, best on the first day, the use of a cathartic is recommended, preferably castor oil, calomel, or a saline cathartic. Fever may call for the external application of cold water and evaporating lotions so long as the skin is hot and dry and the temperature elevated (Sternberg). Later tepid water should be used. A hot mustard foot-bath may be repeated several times during the first day. Cold enemata are recommended. The patient should be protected from cold draughts or sudden lowering of temperature. During the height of the fever antipyrin may be advantageously given. Aconite has been recommended during the first day or two of the disease. Later digitalis may be used, with acetate of potassium or ammonium (Bemis), or sodium bicarbonate in ice-cold water (Sternberg).

The acid secretions would seem to call for the use of alkalies. The following alkaline and antiseptic mixture, proposed by Sternberg, has been largely used:

℞. Sodii bicarbonatis, 10.00.
Hydrargyri chloridi corros., 0.02.
Aquæ puræ, 1000.00.
M. Sig.—Two or three tablespoonfuls every hour; to be given ice-cold.

A weak heart calls for stimulation. Stimulants, as a rule, need not be given before the fourth day, and then they must be administered in small quantities in order not to cause vom-

iting. Champagne, brandy, later milk punch, English ale, and Rhine wine may be used.

During the height of the fever no food is required. With the fall of temperature there is usually a return of the appetite. The diet should then consist of milk in small quantities, possibly with lime-water, and chicken-broth, which may be given every two hours. Excessive vomiting may demand rectal alimentation. The return to the normal diet should be gradual.

CHOLERA (Asiatic Cholera; True Cholera).

Definition: An acute infectious disease, caused by the *spirillum choleræ Asiaticæ*, characterized by onset with diarrhœa and vomiting, later showing great prostration, severe cramps or spasmodic contractions of the muscles, with characteristic stools, resembling rice-water, and a cyanotic appearance of the skin.

History: In the southern part of Bengal cholera is endemic. From this region epidemics of cholera have invaded Asia, Africa, Europe, and America. Cholera was described in India several centuries before Christ, by Charaka (Macnamara). Koch believes, however, that true cholera was not endemic in India before 1817, although epidemics of cholera, or a disease resembling cholera, are recorded as early as 1543.

Cholera first reached the United States in December, 1832, by way of Quebec and New York. The disease appeared again in 1835–6; 1848; 1849; 1854; 1866–7 and 1873. Since then emigrants from Europe have brought cholera to America, but quarantine has prevented the disease becoming epidemic.

The *spirillum choleræ Asiaticæ* was discovered by Koch (1884).

Etiology: Asiatic cholera is now generally recognized as a water-borne disease (Hart), due to the *spirillum choleræ Asiaticæ*, the "comma bacillus" of Koch. The spread of the disease is favored by bad hygiene, especially poor sewerage. Cholera is contagious through the ingestion of the excreta of infected cases. The disease shows a preference for age, and for

individuals debilitated by disease or intemperance. The river population, those who work and live on the water, are predisposed to infection. Epidemics of cholera avoid cold weather.

Cholera—symptomatology: *Incubation*, one to five days. Often cholera *begins with diarrhœa*, sometimes accompanied by *vomiting*, frequently coming on during the night. There are pain in the abdomen, headache, and depression. These symptoms increase in severity. Diarrhœa may be severe in the beginning, with pain and tenesmus. The patient suffers *cramps in the calves of the legs*, later in the arms and abdomen. There is great prostration. Vomiting may be more or less continuous. The *stools*, at first muco-purulent and stained with bile, early assume the rice-water character. The *skin is cyanotic and cold.* There are extreme *anxiety, thirst*, sometimes *heart-failure*. These symptoms continue from a few hours to a day. Should the patient survive this stage, a *reaction* sets in, the cyanosis disappears, the skin becomes warm, the diarrhœa improves, and the prostration is relieved. There is always danger of relapse.

Cases of cholera, showing various degrees of severity, have been called *choleraic diarrhœa*, *cholerine*, and *cholera gravis.*

Diagnosis: The knowledge of the *prevalence of the disease*, or of *exposure to the possibility of infection*, is of value in diagnosis, which can be made positive by finding the *spirillum choleræ Asiaticæ in the stools* in a case presenting the *symptoms* of cholera, which are indicative of *intense intoxication.*

Bacteriological examination: A hanging-drop or cover-glass preparation made from the suspected excreta may reveal the presence of spirilla. The cholera spirillum is motile, decolorized by Gram's method, and stains with the ordinary dyes, probably best with a solution of fuchsin. Upon the surface of diluted bouillon in the incubator colonies appear in ten to twelve hours as a wrinkled film. Gelatin in plates, Petri dishes, or tubes, may be inoculated both from the excreta and from the bouillon cultures. The gelatin begins to be fluidified in a day, and presents under the lens an appearance as if the surface were strewn with glass. The gelatin tubes show a

distinct funnel-shaped depression, with the apex downward, from the fluidification of the gelatin. The so-called "cholera red" or indol reaction may be obtained by adding to bouillon cultures that have been in the incubator ten to twelve hours, or to gelatin cultures in which fluidification has occurred, pure sulphuric acid. A reddish-violet or purplish-red color quickly appears. Cultures in litmus bouillon, made in the incubator, show decolorization within a day.

Pfeiffer has shown that when a trace of cholera serum is added to a culture of cholera spirillum and injected into the peritoneal cavity of a guinea-pig, or when the spirilla are injected into immunized guinea-pigs, the cholera vibrios are quickly destroyed. This is the so-called Pfeiffer phenomenon.

Blood-test: The blood of cholera patients causes paralysis and agglutination of the cholera spirilla (see Typhoid Fever). This method promises to be of value both in the diagnosis of cholera and in the differentiation of the cholera spirillum from simulating organisms.

The **prognosis** of cholera, which should always be guarded, depends largely upon the gravity of the symptoms. Complication with pregnancy, abortion, pneumonia, or typhoid fever, makes the outlook more grave.

Prophylaxis demands quarantine at sea of infected individuals, the destruction of the stools in all cases, best by heat, and the abandonment of contaminated water-supplies. Water may be safely used if thoroughly boiled. The patient must be isolated and all articles that come in contact with him should be sterilized.

Cholera—treatment: We know no specific. "We may look for advance in this direction, above all, to modern bacteriology" (Rumpf). The mortality was decreased in a series of 193 cases of cholera treated in Japan, by Nahagawa, with Kitasato's cholera antitoxin. Klebs's anticholerin has been tried with results that would seem to justify a further trial of the remedy.

Cases of cholera without marked symptoms need little or no treatment, aside from prophylactic measures. Diarrhœa calls for rest in bed and the use of opium.

In cases threatened with cyanosis, Reiche injected hypo-

dermically the fluid extract of opium with good results. The intestinal canal should be emptied with castor oil, or possibly better with calomel, which has the advantage that it may at the same time exert some antiseptic influence. As a rule purgation may be continued only a day or two. Cantani (1870) introduced the use of tannic acid enteroclysis. The intestine is irrigated several times a day with one or two quarts of a 1 per cent. solution of tannic acid at 104° F. This method has been modified by v. Genersich, in what he calls diaclysmus, using from five to fifteen quarts of a 0.1 per cent. to 0.2 per cent. solution of tannic acid at about 104° F. The fluid is gradually passed per rectum until there occurs copious vomiting of the irrigating fluid. Remarkable results are claimed for this method, which would seem to be justifiable in very severe cases.

Vomiting may be controlled by cocaine, or better by morphine hypodermatically.

Elimination by the skin, as well as warmth, may be secured by the use of the warm bath, the temperature of which may be increased to 113° F. for fifteen minutes. Three or four ounces of mustard may be added to the water. Should the pulse not show improvement or should syncope supervene, the bath must be discontinued. In most cases the bath does good. Drink in the form of hot or cold water should not be denied the patient. Alcohol in small quantities may do some good, but in large quantities acts unfavorably.

Evidence of cardiac weakness calls for subcutaneous or intravenous infusions of normal salt solution, 0.6 per cent., or the use of camphor in oil, 1 : 8 or 1 : 10, internally or hypodermatically.

CHOLERA MORBUS (**Cholera Nostras**; **Cholera Infantum**).

Definition: An acute infectious disease, occurring especially during the summer in temperate climates, characterized by diarrhœa and in severe cases presenting symptoms identical with those of true cholera.

Etiology: The disease is due largely to the absorption from the alimentary canal of toxic substances, the result of the

action of bacteria. A number of micro-organisms have been isolated from the stools. Among other bacteria, spirilli have been found, but *not* the spirillum choleræ Asiaticæ. Indiscretions in diet are predisposing causes. The disease shows a preference for summer-time, in temperate regions.

Cholera morbus—symptomatology: The symptoms vary from a simple diarrhœa to severe diarrhœa, sometimes with rice-water stools, vomiting, cramps, cyanosis, collapse, possibly death.

Diagnosis: Cholera morbus is to be differentiated especially from true cholera (Asiatic cholera). Young children seem to be more susceptible to cholera morbus than to cholera Asiatica. The persistence of normal stools in severe cases would point to cholera morbus rather than to cholera Asiatica, in which we usually find rice-water discharges. Cholera morbus may so closely resemble true cholera that the differential diagnosis can be made only by bacteriological methods (see Cholera).

Cases may simulate poisoning by arsenic, solanine, and colchicine, when the differential diagnosis may be made by a chemical examination of the contents of the stomach.

Prognosis: Usually good. The mortality is greatest in children and among the aged, invalids, and intemperate.

Cholera morbus—treatment: Offending material should be removed from the alimentary canal. Material remaining in the stomach may be removed by lavage. Usually the material has passed into the intestine before the patient is seen by the physician, when it may be removed by the administration of castor oil, or calomel, gr. iij for an adult, gr. $\frac{1}{6}$–j for children, in one dose or repeated. In cases of persistent vomiting it may be prudent to wash out the intestine with water, to which may be added soap, castor oil, sweet oil, glycerin, ʒiij–vj, or tannin, 1:1000, best by means of the rectal tube. Obstinate vomiting may sometimes be relieved by enemata of chamomile tea, 80 c.c. to 100 c.c., containing tincture of opium, gtt. v (Liebermeister). Vomiting may usually be stopped by swallowing pieces of ice or by the administration of chloral.

In general the treatment is the same as for true Asiatic

cholera (see Cholera). Severe cases may call for salt water infusion.

ANTHRAX (Malignant Pustule; Carbuncle; Wool-sorters' Disease; Splenic Fever; Milzbrand (German); Charbon (French)).

Definition: An acute infectious disease, caused by the *bacillus anthracis*, occurring among animals, especially cattle and sheep, and occasionally in man through accidental inoculation.

Etiology: The specific infectious agent is the *bacillus anthracis*. The bacillus anthracis—Milzbrand bacillus (German), Bacteridie du charbon (French)—is 1–1.25μ broad and 5–20μ long, sometimes growing into long filaments in favorable culture-soil. The ends of the bacilli are concave, so that when joined together, end to end, there is a distinct lenticular interspace between the bacilli. The bacillus is non-motile, forms spores, stains with the usual dyes and by Gram's method, and grows upon the usual culture-media.

Gelatin is liquefied. The bacillus does not seem to be strictly aërobic, since a growth takes place all along the line of inoculation in stick-culture. The spores are very resistant to drying, and may be preserved in a dry condition for years without losing their vitality or virulence. A dry temperature of 140° C. will kill them in three hours (Koch and Wolffhügel); or moist heat at the boiling-point, 100° C., in four minutes (Sternberg). The bacilli, in the absence of spores, may be destroyed by a temperature of 54° C. in ten minutes (Chauveau).

When ingested the bacilli are killed by the gastric juice; but when the spores are ingested they resist the action of the gastric juice and almost invariably cause infection.

The action of the bacillus is due largely to a toxin. Martin made a chemical study of filtered cultures of the anthrax bacillus and found: (1) protoalbumose, deuteroalbumose, and a trace of peptone; (2) an alkaloid; and (3) small quantities of leucin and tyrosin.

Cattle and sheep are infected by the ingestion of spores. Spores are not formed in the body, but only during the saprophytic existence of the organism. The soil becomes infected largely through the discharges of infected animals.

Animals or men (wool-sorters' disease) may be infected through the respiration of air containing anthrax spores suspended in the form of dust. Exceptionally infection may pass from the mother to the fœtus, possibly through some lesion of the placenta.

Man is infected chiefly through contact with diseased animals, either alive or dead. Thus anthrax is found most frequently among butchers, liverymen, shepherds, tanners, wool-sorters, glue-makers, etc. Insects have been accused of spreading the infection, and the disease has been actually produced by inoculation with the stomach, legs, and feelers of carnivorous flies (Bollinger, Raimbert, and Davaine).

Anthrax—symptomatology: Two general clinical forms of anthrax are recognized: (1) *external anthrax*, including malignant pustule and anthrax œdema; (2) *internal anthrax*, including pulmonary and intestinal infection.

Malignant pustule appears especially on the hands, arms, or face, *surfaces most exposed to infection*. Within a few hours after exposure there are *itching* and *uneasiness*, sometimes *tickling*, *burning*, *stinging*, at the point of inoculation, and soon there appears a small *papule*, which later becomes a *vesicle*. The vesicle bursts, discharging a bloody fluid, presenting the appearance of a red papule with a reddish-brown or black central crust. In mild cases the vesicle may dry up and disappear in a few days. In severe cases the *inflammation and induration become extensive*, the inflammation *involving neighboring lymphatics*. At first there is fever; later the temperature becomes less elevated, often subnormal. The case may end in death in three to five days. Recovery is possible.

Anthrax œdema is characterized by extensive œdema of the eyelids, head, hand, and arm, resulting in gangrene. The papilla and vesicle are absent.

The **pulmonary form** *of anthrax* is commonly known as *wool-sorters' disease* and *rag-pickers' disease*. The infection probably takes place from *inhalation* of dust containing *anthrax bacilli or spores*. The attack comes on with *chill*, *prostration*, *pains* in the back and legs, and *fever* (102°–103° F.). The *pulse* is rapid and feeble. *Rapid breathing* and *pain in*

the chest are prominent symptoms. Death may close the scene within twenty-four hours.

The **intestinal form** *of anthrax* occurs usually through the ingestion of infected milk. Wool-sorters' disease sometimes shows affection of the intestine, probably through swallowing dust ladened with anthrax bacilli. The attack begins with a *chill*, with later *vomiting, diarrhœa, fever, pains* in the back and legs. There may be dyspnœa, cyanosis, restlessness, anxiety, and even convulsions and death. There is *enlargement of the spleen;* sometimes hemorrhage from mucous membranes. The skin may show petechiæ or phlegmonous inflammation.

Affection of the brain is rare.

Anthrax—diagnosis: Knowledge of the *occupation* of the individual, in a suspicious case, is an aid in diagnosis. The *bacillus anthracis* may be found in the *pustules;* later in the blood. The bacillus can be separated by inoculation, best of a mouse or guinea-pig. In *intestinal or pulmonary anthrax* the bacilli may be found in the fæces or sputum, respectively, before they appear in the blood.

Prognosis: Grave. Usually good results may be secured in cases of external anthrax that come under treatment early. The outlook is bad in late cases of external anthrax and in all cases of internal anthrax.

Prophylaxis calls for the avoidance of the cause (see etiology). Peterman (1892) injected into the veins of a susceptible animal large quantities of a culture of the anthrax bacillus in ox-serum filtered through porcelain, and thus obtained temporary immunity lasting not longer than a month or two.

Anthrax—treatment: The point of inoculation should be treated with caustics or the cautery. The pustule may be excised, or incised, and dressed with strong antiseptics. Menthol, 2 per cent. solution in alcohol, applied on a gauze strip, with which the cavity may be packed after cleansing as thoroughly as possible, has been found very effective (Braun). The cavity is packed with the saturated gauze, then covered over air tight, and a compress applied. The gauze is left in twenty-four to forty-eight hours. Subcutaneous injections of solutions of bichloride of mercury or carbolic acid around

the pustule, repeated two or three times a day, are recommended.

In internal anthrax active purgation may be resorted to, and quinine is recommended; but treatment is of little avail.

TETANUS (Lockjaw; Trismus; Opisthotonos; Wundstarrkrampf (German)).

Definition: An acute infectious disease, caused by the *tetanus bacillus*, characterized by tonic spasm of certain muscles, marked by trismus and opisthotonos.

Etiology: Due to infection by the *bacillus tetani*. The tetanus bacillus appears to be a widely distributed microorganism in the superficial layers of the soil in temperate and especially in tropical regions (Sternberg). Inoculation occurs chiefly through traumatism, especially wounds, however slight, caused by splinters or nails contaminated with earth or manure. The tetanus bacillus is attenuated by exposure to oxygen and sunlight, and the virulence is increased by passage through the intestines of animals. The more virulent tetanus bacilli are found in the superficial soil that comes in contact with the dung of animals.

Asepsis and antisepsis has diminished the number of cases of tetanus after surgical operations. Not infrequently tetanus occurs after lacerated wounds. The wound through which the tetanus bacillus gains entrance to the body may heal before the disease is recognized, constituting cryptogenetic or "idiopathic" tetanus. So-called idiopathic tetanus is much less frequent than tetanus neonatorum. Other varieties of tetanus are puerperal, rheumatic, and traumatic.

The spores of the tetanus bacillus are very resistant. Henrijean, quoted by Park, caused tetanus experimentally in an animal by inoculation with a piece of splinter which eleven years before had caused the disease.

The symptoms of tetanus are due to the toxins of the tetanus bacillus rather than to the bacillus itself. Mixed infection is common.

Tetanus—symptomatology: *Incubation* varies from one to twenty-two days, usually one to two weeks. The symptoms of tetanus are due chiefly to a poison (toxin) produced by the

tetanus bacillus. From what has been said about the etiology it is not strange that the wounds through which the tetanus bacillus gains entrance to the body are usually situated on those parts of the body which come most frequently in contact with the earth—the feet and hands.

The patient usually first complains of stiffness of the muscles of the neck and jaw. Sometimes the first symptom is a *spasm of the muscles near the point of inoculation.* With the spasm there is *pain.* The *stiffness of the muscles of the neck and jaw* extends so as to prevent opening the mouth (*trismus, lockjaw*); and to cause *retraction of the head*, sometimes complete opisthotonos, arching of the body backward; rarely emprosthotonos, arching of the body forward; or pleurothotonos, arching of the body to one side. The spasms are constant, except during sleep or narcosis (chloral, opium, and chloroform).

There is *difficulty in swallowing.* Affection of the muscles of the *face* causes the sardonic grin, *risus sardonicus*, described by Hippocrates. There may not be fever. There is *free perspiration.* There may be *difficulty of breathing and cyanosis.*

Diagnosis: *Stiffness* of the muscles of the neck and jaw, especially following *trauma* with liability of infection with the *tetanus bacillus*, should lead to suspicion of tetanus.

The *differential diagnosis* concerns strychnine-poisoning, which shows no period of incubation, and in which the muscles of the extremities are most frequently affected; and hydrophobia, in which there is early difficulty in respiration, from attempts to swallow.

Tetanus—prognosis: Always grave. Rarely cases may show only stiffness of the muscles of the neck and jaw, but the diagnosis of such cases is doubtful. In severe cases death may occur within two or three days. As a rule death takes place in eight to twelve days, or recovery in three to six weeks. Death usually occurs through spasm of the muscles of respiration or through heart-failure.

Tetanus—prophylaxis: Infected wounds should be treated antiseptically. Nails or splinters removed from wounds should be examined for the bacillus tetani; and if this is

found, excision of the infiltrated area or the amputation of a member may be considered.

In cases in which the spasm first appears in the muscles near the point of inoculation, resection of the nerve leading to the area of the wound has in some cases appeared to prevent the development of the disease.

Tetanus—treatment: Most is promised by the *antitoxin treatment*. The subcutaneous use of the tetanus antitoxin has given a mortality of 50 per cent. or less. Better results have been secured by the intracerebral injection of the antitoxin.

It is believed that the tetanus toxin reaches the brain and cord through the nerves and blood. Knorr made an emulsion of the cerebrum of a guinea-pig, to which he added tetanus toxin. The mixture was then centrifugalized. Thus there was secured a precipitate, consisting of the cerebral matter, evidently in intimate association with the toxin, since the upper layer of fluid was found to contain none of the toxin.

When the tetanus toxin is injected into the brain-substance the union between the poison and the cerebral matter is so prompt that the action of the toxin may be limited to certain groups of cells (Roux and Borrel). Different symptoms are produced by the injection of the toxin into various parts of the brain. Animals, whose blood shows the presence of the antitoxin of tetanus, may succumb to intracranial injections of the toxin. Antitetanic serum is much more effective when injected into the brain than when used subcutaneously.

The antitoxin is of little value in treatment after the development of symptoms. Such treatment is better in subacute than acute cases. In all cases, 20 to 50 c.c. of the antitoxin should be given as early as possible.

In tetanus the phagocytes not only destroy the tetanus bacilli, as far as they are able, but they also absorb the toxin. Baccelli has secured good results by the subcutaneous injection of carbolic acid, 2 per cent. solution, beginning with grs. iij in the twenty-four hours and increasing to gr. vj–ix in the twenty-four hours. There is remarkable tolerance to carbolic acid in tetanus.

General treatment: The patient should be kept quiet, and secluded from light and noise as well as from unnecessary visitors. Feeding should not be neglected. The diet should be light and nutritious, and if necessary may be given per rectum.

Various remedies have been used, among them the bromides, Indian hemp, chloral, opium, chloroform, atropine, calabar bean, curare, and carbolic acid.

HYDROPHOBIA (**Rabies** (**Latin**); **Wuth, Hundswuth, Tollwuth** (**German**); **La Rage** (**French**)).

Definition: An acute infectious disease, communicated to man from the lower animals, especially the dog ("mad dog"), by inoculation, usually through bites, and characterized in man by fear of water, or rather by inability to swallow, a symptom that is absent in animals.

Etiology: The disease is found in the dog, fox, wolf, cat, and skunk, and may be communicated to other animals or to man by *inoculation*. Hydrophobia may be communicated at any time in the course of the disease, even during the period of incubation.

Hydrophobia — symptomatology: *Incubation* usually lasts from six weeks to two months, but is very variable.

The *premonitory stage* is dominated by *disturbances in the psychical sphere*. There are depression and melancholia, headache and anorexia, insomnia and irritability, increased sensibility, and a feeling of impending danger. The *larynx* is injected, and there may be some difficulty in *swallowing*. The *point of inoculation* may show irritation, pain, or numbness.

The *stage of excitement*, which lasts from a day and a half to three days, is marked by great *hyperæsthesia*, *excitability*, *restlessness*, and *inability to swallow*. Water or liquid food is more dreaded because it more readily suggests the act of swallowing. *Mania*, or general convulsions, may be present. Sometimes there is satyriasis or nymphomania. There is usually some fever, 100° to 103° F.

Paralytic stage: Gradually the spasms disappear, and the

patient becomes quiet, and later unconscious. The heart's action becomes feeble, and death by syncope may occur in from six to eighteen hours.

The **diagnosis** of hydrophobia is easy in the presence of typical symptoms, especially spasm of the muscles of deglutition and respiration, and the history of exposure to infection, most commonly a dog-bite.

The *differential diagnosis* concerns especially lyssophobia, hysteria, Landry's paralysis, tetanus, and uræmia.

Prognosis: Bad. Recoveries from hydrophobia have been claimed; but with our present knowledge, the positive demonstration of the disease would be difficult in cases that recover. Dogs have recovered from rabies. Much is claimed for the treatment recommended by Pasteur.

Hydrophobia—prophylaxis: Rabid animals should be killed and all dogs should be muzzled or confined.

Bites of animals, especially of those suffering from rabies, should be thoroughly cauterized and kept open. Keirle gives a mortality of 30 per cent. following cauterization, against 80 per cent. in cases that were not cauterized. Where cauterization may not be resorted to at once, it may be advantageous to ligate above the wound; to suck the wound or apply cups, or open up the wound, or even to resort to amputation. In all cases it is best to make free use of antisepsis.

Where practicable, the individual should receive the prophylactic inoculations recommended by Pasteur.

Pasteur found the virus of hydrophobia located in the central nervous system, especially the spinal cord. Inoculation from rabbit to rabbit increased the virulence and decreased the period of incubation. The virus used as a standard will cause the symptoms of hydrophobia after an incubation of seven days. The virus is attenuated by desiccating the spinal cords in sterilized glass jars containing caustic potash. After two weeks' desiccation, the spinal cord is perfectly innocuous. Beginning with the injection of an emulsion of such a non-virulent cord, successive injections are made of the emulsions of more virulent cords—that is, cords that have not been desiccated so long—until the individual is able to receive an injection of the emulsion of a cord that has been desiccated

only five days. At this point the greatest protection is secured.

Hydrophobia—treatment: The patient should be kept in a darkened room and visitors excluded. The spasms may be allayed by inhalations of chloroform and the use of morphine hypodermatically. The patient should be fed. Liquid food may sometimes be given after the throat is cocainized. If necessary, food may be given per rectum.

DENGUE (Break-bone Fever).

Definition: An acute infectious disease of short duration, characterized by severe pains in the head, eyeballs, and joints; inflammation of exposed mucous surfaces, swollen salivary glands, and a peculiar eruption. The disease often occurs in epidemic form, and is in some places endemic, as in Calcutta.

The **etiology** is not clear. McLaughlin (1886) claimed to find micrococci in the blood in cases of dengue, but the observation lacks confirmation.

Dengue—symptomatology: Incubation, two to five days. The onset is sudden, with chilly sensations, headache, congested conjunctivæ, and pains in the eyeballs, muscles, and joints. The mucous membranes exposed to the air become inflamed. There are sore throat and swelling of the submaxillary glands. The fever rises gradually to 103° to 107° F.; pulse 100 to 120; respiration hurried. A scarlatiniform rash appears within one or two days, usually first upon the face, sometimes on the chest, back, abdomen, and knees, and lasts about a day. From three to four days later the terminal rash appears, usually first on the palms of the hands, sometimes followed by desquamation. This rash may be so slight as to be scarcely observed, or so severe as to cause ecchymoses. There may be some fever. Convalescence is tedious. The severity of individual symptoms varies in different epidemics, as well as in different cases.

Diagnosis: At first dengue may resemble scarlatina or rheumatism, but later the differentiation is easy. The resemblance between dengue and mild cases of yellow fever is more marked. Suspicious cases should be isolated until the differ-

entiation is absolute. Dengue should be differentiated also from influenza, typhoid fever, and malaria.

Prognosis: Adults rarely die. Death occasionally occurs through some complication, such as septicæmia following abortion. Children may suffer convulsions and death.

Dengue—treatment: The patient should have good hygienic surroundings, thorough ventilation, and isolation. The intestinal canal should be cleansed, best with calomel, rhubarb, or colocynth; but the use of active purgation or emesis is uncalled for. Temperature above 105° F. calls for hydrotherapy, the cold bath, or cold sponging. Tincture of belladonna, gtt. x–xv, gives great relief. When there is pain opium may be given, especially to secure sleep. Opium is probably best given in the form of Dover's powder.

Complications call for appropriate treatment.

PLAGUE (Bubonic Plague; the Pest).

Definition: An infectious disease, due to a specific bacillus, characterized by swelling of the inguinal and other lymphatic glands (buboes), often with the appearance of carbuncles and hemorrhages.

History: The disease probably existed before the beginning of the Christian era, but the first reliable account is of an epidemic in Constantinople, 542 A. D. The bacillus was discovered by Kitasato and Yersin, during an epidemic in China, in 1894, when the disease prevailed especially in Hongkong and Canton. The plague has never appeared in America.

Etiology: The bacillus of bubonic plague, the *bacillus pestis* of Kitasato, is found in all cases of plague, and has been proven by inoculation of pure cultures to be the specific cause of the disease. Bad hygienic surroundings are supposed to be predisposing causes.

Plague—symptomatology: Incubation, two to eight days. Usually the symptoms come on rather suddenly, with lassitude, loss of strength, mental anxiety, sometimes with headache and vertigo, pain in the back and limbs, fever, and delirium. There is an invasion of the lymphatics in two or three days, which ends in resolution or suppuration, sometimes

gangrene. Carbuncles, petechiæ, or purpuric spots may appear upon different parts of the body.

Diagnosis: Pains in the regions of the lymphatics, especially the inguinal, with later tenderness, swelling of the glands, and the formation of buboes, with the appearance still later of carbuncles and hemorrhages, stamp the disease. In doubtful cases the bacillus should be isolated.

The plague should be *differentiated* from lymphadenitis due to other causes—tuberculosis, syphilis, typhus fever, and anthrax.

Prognosis: Should be guarded. Death may occur within a few hours. Much depends upon the severity of the symptoms.

Prophylaxis: Calls for proper sanitation, especially regarding sewage and water-supply. Patients should be isolated until at least a month after recovery. The dead should be buried at a depth of three meters, or preferably cremated (Kitasato). The excrement and all articles that come in contact with the patient should be burned or thoroughly sterilized.

Plague—treatment: With the *serum treatment* Yersin, in cases treated with strong serum, had only two deaths in twenty-six cases. Further treatment is *symptomatic.*

ACUTE INFECTIOUS ICTERUS (Acute Febrile Icterus; Weil's Disease).

Definition: An acute infectious disease, characterized by fever, prostration, icterus, and gastro-intestinal disturbances.

Etiology: Probably due to the *bacillus proteus fluorescens* (Jaeger). The disease seems to show a preference for summer and the male sex.

Acute infectious icterus—symptomatology: Prodromata are usually absent. The onset is usually sudden, often with a chill. On the fourth or fifth day the fever may remit and recur in two or three days, lasting eight to ten days. There are intense prostration and marked jaundice, mental dulness, sometimes delirium and coma. The urine, diminished in quantity, contains bile, and in about half the cases albumin and casts, sometimes blood.

Diagnosis: The absence of prodromata, the sudden onset, the remissions, and the duration of the disease are more or less characteristic. The disease should be *differentiated* from simple catarrhal jaundice and from typhoid fever with jaundice.

Prognosis: Usually good. Death has occurred, but recovery is the rule.

Acute infectious icterus—treatment: A milk-diet is best. Active purgation should be avoided. Small doses of calomel or castor-oil may be given early in the disease. Carlsbad water or the Carlsbad salt is used. Irrigation of the large intestine is recommended.

MALTA FEVER (Mediterranean Fever; Rock Fever; Neapolitan Fever).

Definition: An infectious disease of long duration, caused by the *micrococcus melitensis*, characterized by fever, prostration, constipation, relapses, enlargement and softening of the spleen, often by rheumatic or neuralgic pains, sometimes by swelling of the joints and orchitis.

Etiology: The *micrococcus melitensis* (Bruce, 1887) has been proven to be the specific infectious agent, by inoculation of animals and by an accidental inoculation in man. The disease is endemic upon the island of Malta, and appears also in Naples and other Mediterranean ports; is more prevalent during the hot months—May, June, especially July. The disease has appeared in the United States.

Malta fever—symptomatology: *Incubation* six to thirty days, usually about two weeks.

The *early symptoms* are malaise, anorexia, nausea, sometimes vomiting, sleeplessness, epistaxis, coated tongue, congestion of the pharynx, as a rule constipation, sometimes diarrhœa from indiscretion in diet, the stools sometimes containing blood, with enlargement of the spleen and liver, profuse perspiration, sudamina, usually a slight cough with scanty expectoration and moist crepitant râles which last a week or ten days, sometimes a month. The symptoms clear up and the patient apparently enters convalescence. Sooner or later there is a *recurrence of symptoms*. There are considerable pros-

tration and marked weakness. The number of red blood-corpuscles is diminished. Temperature 101°–104° F., and irregular. There are pains in the joints, which show swelling; intercostal neuralgia, sciatica; and orchitis. After some weeks' duration the fever gradually subsides, the number of red blood-corpuscles returns to the normal, the strength improves, and the weight increases.

The symptoms may occur in all grades of severity. In some cases the symptoms may be so slight that only a rise in temperature will be noticed. The disease usually lasts about two or three months.

Diagnosis: Malta fever is to be differentiated especially from typhoid fever, which it often simulates so closely as to be recognized only by the clumping of the micrococcus melitensis upon the addition of the serum, should the case be Malta fever; or by the paralysis and clumping of typhoid bacilli, in the blood-test, in cases of typhoid fever. Malaria may be ruled out by a search for the plasmodium.

Prognosis: The mortality is about 2 per cent.

Malta fever—prophylaxis: If possible, the region of the Mediterranean should be avoided, especially during the hot months. Where this is not possible special attention should be paid to hygiene, especially with regard to sanitation and personal cleanliness. Fatigue and intemperance should be avoided.

Malta fever—treatment: The diet, consisting largely of milk, eggs, beef-tea, and brandy, must be continued for several weeks. Fresh lemonade or lime-juice should be added, to prevent scurvy. After the temperature has remained normal two weeks the patient may return to ordinary diet.

The treatment is symptomatic. High temperature calls for the cold bath, which must be repeated whenever the temperature reaches 103° F.

MILIARY FEVER (Sweating Fever).

Definition: An infectious disease, occurring especially in France, Italy, Germany, and Austria, characterized by fever, profuse sweating and a miliary eruption of vesicles.

History: The disease was first described in London, 1485, as sudor Anglicus; Leipsic, 1652; France, Montbeliard, 1712, and Abbeville, 1718.

The **etiology** is obscure.

Miliary fever—symptomatology: There may or may not be prodromata, lassitude, anorexia, and headache. Perspiration is profuse and persistent. There is great thirst; the mouth is dry; the tongue is coated. Usually there is constipation. About the third day, as a rule, the miliary eruption appears, preceded by a pricking sensation and itching of the skin, first as papules, which later become vesicles. The eruption lasts two or three days, and the symptoms disappear within a week or ten days from the onset of the disease.

The nervous phenomena are prominent: constriction or oppression in the epigastric region with mental anxiety; palpitation; sometimes cardialgia and constriction of the pharynx. Occasionally there are delirium, less constantly general malaise, fatigue, headache, pains in the joints, vertigo, and insomnia.

Diagnosis: In the presence of an epidemic the diagnosis is usually easy. Miliary fever should be differentiated especially from scarlet fever, puerperal sepsis, and measles.

The **prognosis** varies greatly in different epidemics.

Prophylaxis calls for sanitation, isolation, disinfection.

Treatment is symptomatic.

BERIBERI.

Definition: An infectious disease, occurring especially in tropical and subtropical regions, characterized by motor paresis, beginning in the lower extremities, with œdema and sensory disturbances, visceral disorders, especially of the heart and lungs. The disease is of long duration, and frequently shows acute exacerbations.

Etiology: Various micro-organisms, chiefly *micrococci*, have been described.

Beriberi—symptomatology: The disease shows almost infinite variations and combinations of symptoms. The symptoms most frequently present depend upon paresis, atrophy, numbness, and œdema. There may or may not be fever.

The pulse varies greatly in different cases. Usually there is palpitation. Perspiration may be diminished or absent, or greatly increased.

Diagnosis: Usually easy in regions where the disease is endemic.

Beriberi should be *differentiated* especially from locomotor ataxia, progressive muscular atrophy, paralysis, myelitis, polyneuritis, diseases of the heart, anæmia, malaria, and Bright's disease.

Prognosis: Should be guarded. Mortality varies greatly in different epidemics.

Prophylaxis: Demands isolation, proper attention to hygiene, especially sanitation and disinfection.

Treatment is symptomatic.

GLANDULAR FEVER.

Definition: An acute infectious disease, characterized by adenitis and the absence of eruption.

Etiology: The disease occurs most frequently in childhood, sometimes in infancy, rarely in age. The specific cause is unknown.

Glandular fever—symptomatology: Incubation, five to fifteen days, usually six or seven days. The onset is sudden with malaise, nausea, sometimes vomiting. The tongue is coated. Temperature 101°–103° F. Constipation is the rule. The anterior cervical glands are most frequently affected, usually first on the left side. There is apparently stiffness of the neck, since movement causes pain. The glands show enlargement about the second or third day, when the temperature is highest. Uncomplicated cases do not show suppuration. Usually the liver is enlarged. Enlargement of the spleen is found in about half the cases. The beginning of convalescence is usually marked by the passage of thin green stools containing mucus. The glands begin to diminish in size from two to five days after they begin to swell. As a rule the fever and symptoms continue five to ten days, sometimes as long as two weeks, when there is a successive involvement of different groups of glands. The patient is depressed and anæmic. Convalescence requires one or two months.

Diagnosis: Tonsilitis and pharyngitis should be excluded. Glandular fever should be differentiated especially from irregular cases of rubella and mumps.

Prognosis: Good. Death may occur in the case of delicate children.

Treatment is symptomatic.

SIMPLE CONTINUED FEVER.

Definition: Cases characterized by an elevation of temperature, more or less continuous, which may not be classified under any of the known diseases.

Etiology: Probably due to a number of causes. The diagnosis of "simple continued fever" frequently arises from a failure to recognize the true nature of the disease.

Symptom: The only characteristic is the elevation of temperature, which may last from a few days to a few months.

Diagnosis: Other diseases should be ruled out, especially tuberculosis, typhoid fever, malaria, and intestinal ptomain-poisoning.

Treatment symptomatic. Persistent cases call for change of residence.

HAY FEVER (Autumnal Catarrh; Catarrhus Æstivus; Rhinitis Hyperæsthetica; Hay Asthma; June Cold; Summer Catarrh.)

The disease occurs most frequently in the fall, and is marked by catarrh of the upper air-passages, especially of the nose, with coryza, sometimes inflammation of the eyes, conjunctivitis, and lachrymation.

Etiology: Hay fever has for a long time been ascribed to an irritability of the nervous system, and it has been observed that the attacks are apparently caused most frequently by the pollen of ragweed (Ambrosia artemesifolia) and golden rod (Solidago odora); more rarely by wheat, barley, oats, rye, and Indian corn. Dust and the odor of animals and flowers sometimes cause the disease. The disease is probably due to some *micro-organism* that finds a favorable soil in the pollen. Often there is hyperæsthesia of the nasal mucous membrane.

Symptoms: Hay fever is most frequent in middle life, but

infancy and old age are not exempt. The disease shows a peculiar *periodicity*, in that the attacks recur each year upon about the same day, sometimes at the same hour. There are noticed early tickling and irritation of the conjunctivæ and of the mucous membrane of the upper respiratory passages, especially of the nose. Soon there are *sneezing*, *coryza*, and *lachrymation*. There may be two or three degrees of fever and some increase of the pulse-rate. With the local symptoms there is more or less *malaise and prostration*. Sometimes there is *pain* in the muscles, eyes, and occipital region. The general symptoms seem to be due to a *toxæmia*. Sometimes early, usually after the disease has existed two or three years, *asthmatic attacks* assume prominence. These usually appear late in the season, but may begin early in the attack. The attacks usually cease after a few hard frosts.

Diagnosis: The coryza, often with lachrymation, sometimes with asthma, comes on suddenly, about the same time on succeeding years. *Differential diagnosis* has to do chiefly with acute nasal catarrh, influenza, and spasmodic asthma.

Prognosis: Hay fever is not a fatal malady. Almost all cases may be relieved, many may be cured, but some cases persistently recur year after year.

Hay fever—treatment: A change of place of residence, especially to a cooler climate, will relieve many cases. Sometimes it is only necessary to take a trip at the time of the expected attack. Some cases may be benefited by tonics.

In the way of *palliative treatment* most relief is afforded by opium and belladonna, or morphine and atropine, but these remedies should not be used indiscriminately as a routine treatment. The local application of cocaine may give great but only temporary relief. Nasal spurs or adenoids should be removed, which is sometimes followed by a cure of the hay fever. Sensitive areas, which are most frequently found upon the nasal septum, may sometimes be cured by cauterization, after which the hay fever may disappear.

Probably the best single remedy is *arsenic*, Fowler's solution, gtt. ij–v three times a day up to tolerance. Quinine may be given, gr. v, morning and evening. A boracic acid oint-

ment in vaseline, 5 per cent., is useful. Conjunctivitis is relieved by the instillation of cocaine, 4 per cent. solution, or morphine, 1-2 per cent. solution. Chlorate of potassium may be given internally, a teaspoonful of the saturated solution every two hours. The asthma is sometimes relieved by chloral, gr. v, or the iodides. Headache and fever are relieved by phenacetin and the salicylates.

ACTINOMYCOSIS (Big Jaw; Swelled Head; Holzzunge, Knochenkrebs, Kinnebeule (German)).

Definition: A disease, found especially in cattle, sometimes in man, caused by the *ray fungus, actinomyces.*

Etiology: In the pus or granulations the fungus appears as whitish, more often yellowish, granules, which under the microscope are seen to consist of threads radiating from a centre and ending in club-shaped extremities. Bostroem would classify the parasite among the polymorphous bacteria, since the masses contain cocci and bacilli, some of which are branched and show club-shaped extremities (Ponfick) (see Tuberculosis).

The organism has been cultivated outside of the body, and inoculation-experiments upon animals have been successful.

The disease may occur in man by direct transmission from infected animals or from foreign bodies, especially cereal grains with sharp extremities, more rarely isinglass, splinters, etc., which may contain growths of the parasite. Infection may occur through carious teeth, and Ponfick has reported infection from barbers' utensils.

In *man*, actinomycosis is found most frequently in the head (jaw, tongue), neck, air-passages (lungs), alimentary canal (small intestine), and skin.

Actinomycosis — symptomatology: The infection runs a *chronic course.* The symptoms, at first obscure, increase insidiously, and show variations according to the location of the actinomycotic process. As a rule, wherever the process develops there is the formation of *granulation-tissue, abscesses, and fistulæ.* The infiltration is peculiar, and has been described by Ponfick as "tough." *Bones*, when attacked, are

expanded and eroded. Affection of the *lungs* may cause cough, expectoration, irregular fever, emaciation, night-sweats, and the formation of cavities closely resembling pulmonary tuberculosis, and life may be terminated by tuberculosis or amyloid degeneration.

In the *intestine* the process is slow, permitting protection of the general peritoneal cavity by the adhesion of coils of intestine. Frequently the first suspicion of the disease may be afforded and verified by the discharge of the *peculiar granular pus containing the parasite,* from a sinus which may be in the lumbar, more rarely in the gluteal or perineal, region; sometimes in some other part of the abdominal wall, communicating with the intestine or bladder.

Diagnosis, to be absolute, depends upon the detection of the parasite, *ray fungus,* which not infrequently is to be found in granular pus discharged from a sinus; sometimes in the sputum, in cases of involvement of the air-passages (lung). Suspicion may be aroused by the *insidious onset* and *chronic course* of the infection, the presence of *granulation-tissue, fistulæ, and abscesses.*

Karlowski gives, as a point in differential diagnosis, that dulness is found below the clavicle in actinomycosis of the lungs, and not at the apex of the lung as in tuberculosis.

Prognosis: Depends upon the location of the process, especially upon the accessibility of the infectious foci to surgical treatment.

Prophylaxis: Care should be taken of the teeth and mouth. Animals should receive good food, not containing thorns. The parasite should be destroyed, best by fire.

Actinomycosis—treatment: Wherever possible the deposits of the fungus should be thoroughly removed or destroyed with the knife or cautery. Where this is not practicable, Ponfick advises repeated injections of bichloride of mercury, 1 : 500. Further treatment is symptomatic.

Karlowski successfully treated a case of pulmonary actinomycosis by incision, resection of a rib, the use of the Paquelin thermo-cautery, and the application of iodoform-gauze. The internal use of iodide of potassium has been recommended in visceral affection, but was found ineffectual

by Poncet in 18 out of 25 cases. The remedy is useful only in the earliest stages.

DISEASES CAUSED BY ANIMAL PARASITES.

MALARIA (Intermittent Fever; Chills and Fever; Ague; Swamp Fever; Marsh Fever; Miasmatic Fever; Wechselfieber (German)).

Etymology: Malaria, from mal'aria (Italian), meaning bad air. It has been suggested that mal aqua would be a better name for the disease.

Definition: An infectious disease, acute or chronic, caused by the hæmatozoön (plasmodium) malariæ; appearing sometimes as a pernicious fever; usually as a fever of intermittent or remittent type; frequently as a chronic cachexia with anæmia and enlargement of the spleen.

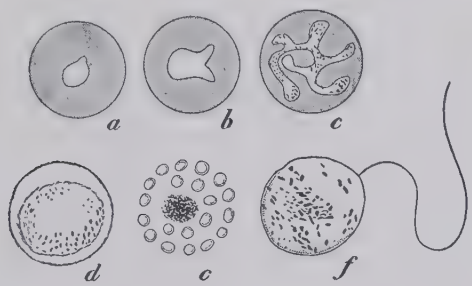
History: Malaria was known in the remotest antiquity. The disease was described by Hippocrates. Celsus and Galen recognized the quotidian, tertian, and quartan types. Pernicious paroxysms were described by Mercatus, toward the end of the sixteenth century. Cinchona bark was introduced into Europe, in the treatment of malaria, by the Countess del Cinchon and her body-physician, Juan del Vega, 1640. The malarial parasite was discovered by Laveran in November, 1880. Golgi described some of the varieties of the parasite found in quartan and tertian types of the disease, 1885–6. Marchiafava and Celli described varieties of the parasite in æstivo-autumnal fever, 1889.

Etiology: The specific cause of malaria is now generally recognized to be the malarial parasite, the *oscillaria malariæ* of Laveran, more commonly known as the *plasmodium malariæ* (Marchiafava and Celli), more properly the *hæmatozoön*, or better the *hæmocytozoön malariæ*. The term *hæmosporidium* has been recommended, but not generally adopted.

The malarial parasite belongs to the *protozoa*, a class of unicellular animals, and to the group *hæmocytozoa*, since it develops within a red blood-corpuscle. There is a difference of opinion as to whether the different forms of malaria (tertian, quartan, and autumnal) are due to the same organism

or to different varieties of the malarial parasite. It is known that certain appearances of the parasite are peculiar to the different forms of malaria.

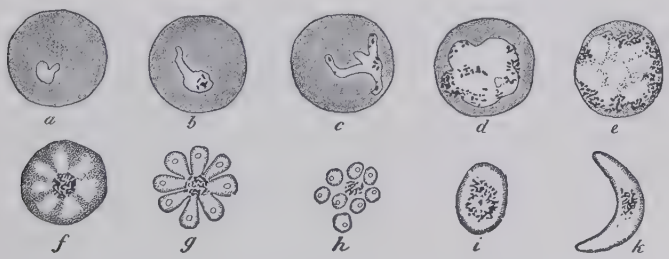
Fig. 15.



Plasmodium malariæ of a febris tertiana in various developmental stages (after Golgi). *a*, first step in development; *b*, *c*, enlarged plasmodia with pseudopods; *d*, plasmodia before the formation of spores—blood-corpuscle decolorized; *e*, formation of spores; *f*, free parasite with flagella.

In the *tertian* form of malaria the parasite (Fig. 15) appears first as a small hyaline amœboid body, becomes pigmented with granules in active motion, and grows to about the size of a

Fig. 16.

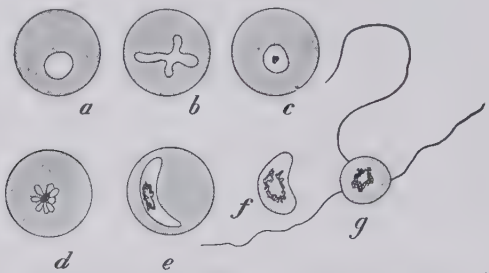


Plasmodium malariæ of a febris quartana in various stages of development (after Golgi). *a*, red blood-corpuscle with a small, non-pigmented plasmodium; *b*, *c*, *d*, *e*, pigmented, variously sized plasmodia inside of red blood-corpuscles; *f*, plasmodium at the commencement of segmentation, with pigment collected in centre; *g*, segmented plasmodium; *h*, plasmodium divided into separate globules; *i*, *k*, two differently shaped, free plasmodia.

red blood-corpuscle. The corpuscle becomes expanded and decolorized. The parasite then breaks up into fifteen or twenty segments (spores).

In the *quartan* fever (Fig. 16) the amœboid movements are slower than in the tertian form, and the granules of pigment are coarser and present less active motion. The corpuscle contracts around the parasite and shows a somewhat

Fig. 17.

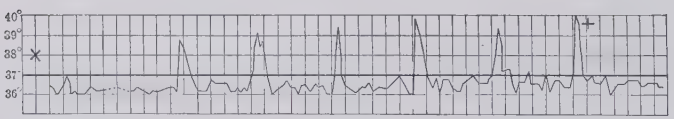


Plasmodium malariæ of a febris quotidiana in various stages of development (after Celli and Sanfelice). *a*, first step in the development; *b*, plasmodium with pseudopods; *c*, plasmodium which has become round and provided with pigment before segmentation; *d*, formation of spores; *e*, intraglobular crescent form; *f*, *g*, free plasmodia.

deeper color. The parasite breaks up into only five or ten segments, arranged in the form of rosettes around a central clump of pigment.

The *æstivo-autumnal* parasite is still smaller, reaching only half the size of a red blood-corpuscle, and presents less pig-

Fig. 18.



Temperature-curve in man after injection of blood from patient affected with malarial (quartan) fever. × 12, noon, injection of four cubic centimetres of blood: + injection of two grammes of muriate of quinine (Baccelli).

ment. The corpuscles become contracted around the parasite, often crenated. After about a week the characteristic crescentic, ovoid, and round bodies appear, containing central clumps of coarse pigment-granules. The round bodies of this

form of the parasite, as well as the full-grown tertian and quartan parasites, may present *flagelli*, which show active movement and may become detached from the corpuscles and appear free in the blood.

MacCallum has shown that the flagelli are the male elements of reproduction.

Malaria has been transmitted by subcutaneous inoculation with blood, the disease appearing in the same form as in the case from which the inoculation was made. It is supposed that infection may also gain entrance to the body through the respiratory tract, but this has not been proven.

Nothing is known of the *life-history* of the parasite outside of the body. Attempts at cultivation upon artificial media have been unsuccessful.

A *peculiarity* of malaria is that it may prevail in a region for an indefinite length of time and suddenly disappear, seemingly without cause, to reappear at some future time. The disease seems to prefer a low, swampy country and to avoid altitude. Often the occupants of the ground floor of dwellings may be attacked, while those in the upper stories escape.

An *exception to the rule* seems to be found in Quetta, India, which is almost 6000 feet above the level of the sea, and upon which some hills reach 12,000 feet above sea-level. Quetta is affected periodically by malaria, especially during September and October (Birch).

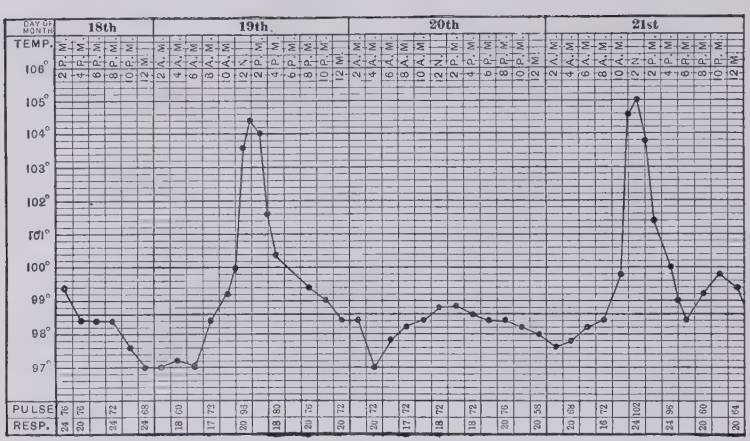
The *mosquito* plays a prominent rôle in carrying the infection, producing the disease in man by direct inoculation.

Grassi believes the species of mosquito that acts most frequently as purveyors of malaria are the following: *anopheles claviges* (Fabr), *culex penicillaris* (Roudani), and *culex hortensis* (Ficalbi).

Malaria—symptomatology: The period of *incubation* is not accurately known, but probably varies from one to two weeks. In cases where malaria has been produced experimentally the incubation has lasted in the *quartan* type from eleven to fifteen days; in the *tertain* type, six to twelve days; and in the *æstivo-autumnal* type, two to five days.

The regular *intermittent* types, tertian and quartan, are characterized by *regularly recurring paroxysms* of *chill* followed

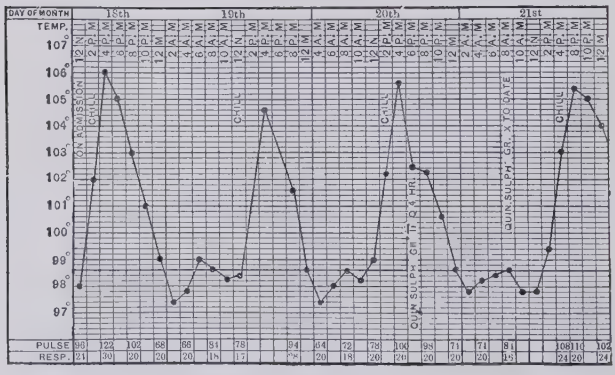
FIG. 19.



Tertian fever (Seguin).

by *fever*, later *sweating*. The paroxysms are often preceded by uneasy sensations, especially in the epigastrium, and some-

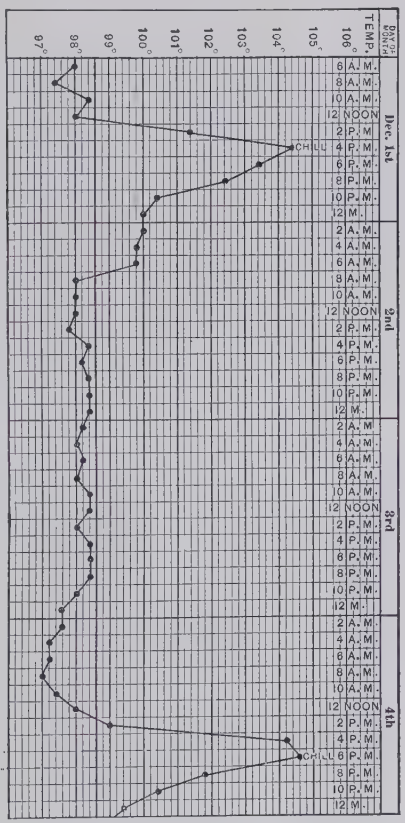
FIG. 20.



Quotidian fever (Seguin).

times by headache. With the onset of the *paroxysm* there are lassitude, headache, sometimes nausea and vomiting, a

slight rise in temperature, and a pronounced chill, the skin becoming cold and blue. The temperature rises, and may reach 105° or 106° F. The pulse is rapid, hard, and non-compressible. There is headache. The chill may last from



Quartan fever (Seguin).

FIG. 21.

ten minutes to an hour or longer. Gradually the temperature of the surface changes from cold to hot, the face is flushed, and the skin reddened. There may be throbbing headache. There is intense thirst. The hot stage lasts from

thirty minutes to three or four hours. Gradually sweating appears, the temperature falls, the headache is relieved, and soon the paroxysm is over. The paroxysm usually lasts ten to twelve hours. *Between paroxysms the individual is apparently well.* In the *tertian* type the paroxysms recur every third day—that is, about forty-eight hours apart. An infection with two sets of tertian parasites may cause daily paroxysms—the *quotidian* fever. In the *quartan* type the interval is about seventy-two hours, the paroxysms occurring every fourth day. A *double quartan infection* may cause paroxysms on two successive days, followed by an intermission of one day. A *triple quartan infection* may cause daily paroxysms—a *quotidian* fever. The disease may disappear spontaneously in ten days or two weeks; but recurrence is frequent. Or the disease may become chronic, to cause *malarial cachexia.*

The symptoms of *æstivo-autumnal fever* are more irregular, the paroxysms usually lasting about twenty hours. Often the onset is without chill, sometimes even without chilly sensations. Frequently the temperature rises and falls slowly, instead of abruptly. Sometimes the fever is more or less continuous, running about 102° to 103° F. During the paroxysms the temperature reaches 105°, sometimes 106° F. Frequently there is jaundice.

Pernicious malaria may occur in the comatose, algid, convulsive, or hemorrhagic form.

Malaria—diagnosis: Usually the *regular recurrence* of *chill, fever*, and *sweating*, with *enlargement of the spleen*, is sufficient to enable the physician to make a correct diagnosis. Doubtful cases call for microscopic examination of the blood for the *parasite;* or the administration of quinine, methylene-blue, or arsenic, as a *therapeutic test.*

Prognosis: Usually the tertian and quartan types have a good prognosis, as have also cases of æstivo-autumnal fever that come under treatment early. The prognosis is not so good in cases showing the symptoms of pernicious malaria, especially when the paroxysms continue forty-eight hours after beginning treatment.

Prophylaxis: In regions where malaria is prevalent the sleeping-apartments should be as far above ground as possible,

and the individual should be protected from the mosquito. It has been suggested that the drinking-water should be boiled. Quinine, in small doses, two or three grains three times a day, affords protection. The inhabitants of non-malarial regions should visit malarial districts only when the disease is least prevalent.

Malaria—treatment: Quinine for malaria is one of the few specifics in medicine. The remedy is best given in round doses just before an expected paroxysm. Gr. xx–xl daily may be continued for three days, then smaller doses given for two or three weeks. Severe cases may call for the administration of quinine hypodermatically or by intravenous injection.

The following are the salts of quinine usually employed, with their percentage of quinine and solubility in water:

Salts of Quinine classified according to the Percentage of the Alkaloid which they contain.

	Quinine.
100 parts of the basic muriate of quinine contain	81.71 per cent.
" " neutral " " "	81.61 "
" " basic lactate " "	78.26 "
" " " hydrobromate " "	76.60 "
" " " sulphate " "	74.31 "
" " " sulphovinate " "	72.16 "
" " neutral lactate " "	62.30 "
" " " hydrobromate " "	60.67 "
" " " sulphate " "	59.12 "
" " " sulphovinate " "	56.25 "

Salts of Quinine classified according to their Solubility in Water (Regnauld and Villejean).

	Water.
1 part of the neutral hydrochlorate of quinine is soluble in	0.96
" " " sulphovinate " " "	0.70
" " " lactate " " "	2.
" " basic sulphovinate " " "	3.30
" " neutral hydrobromate " " "	6.33
" " " sulphate " " "	9.00
" " basic lactate " " "	10.29
" " " hydrochlorate " " "	21.40
" " " hydrobromate " " "	45.02
" " " sulphate " " "	581.00

The salt that contains most quinine and is soluble is superior. Therefore, *quininæ hydrochloras* is best.

The *sulphate of quinine*, which is the salt generally used, is much more soluble in an acid medium, and should, therefore, be prescribed with an acid.

Before quinine came into use cinchona bark was employed. Other derivatives of cinchona, besides quinine, have been recommended: cinchonin, cinchonidin, quinidin, and quinoidin.

Methelyne-blue occupies a position next to quinine in the treatment of malaria, and in some cases is preferable to quinine.

Chronic cases of malaria may be treated with arsenic, best in the form of Fowler's solution.

If the patient resides in a malarial district, he should seek more healthy surroundings. A change of residence is especially important in advanced malarial cachexia.

DYSENTERY (Flux; Ruhr (German)).

Definition: An infection, chiefly of the large intestine, characterized by diarrhœa with tormina and tenesmus.

Etiology: Dysentery is most prevalent in tropical and subtropical countries, although during warm weather it may assume an epidemic form in temperate regions. The *amœba coli* is generally recognized as the specific infectious agent in many cases of tropical dysentery. But this organism is not always found in dysentery. Maggiori (1893) found amœba in only one case in twenty. In the remaining nineteen cases the bacteriological examination revealed the *bacillus coli communis* in large numbers; the proteus vulgaris in most cases; and in some cases the bacillus fluorescens liquefaciens, the staphylococcus pyogenes aureus, staphylococcus pyogenes albus, and the bacillus pyocyaneus. Arnaud (1894), from a study of sixty cases of tropical dysentery, comes to the conclusion that the disease is due largely to a pathogenic variety of the bacillus coli communis. Rectal inoculation with cultures of the colon bacillus produced dysentery in dogs.

It is generally believed that dysentery is disseminated

largely through the medium of impure drinking-water, contaminated by fæcal matter.

Dysentery — symptomatology: The symptoms vary in severity. Preceding the attack there may or may not be diarrhœa. Sooner or later the attack comes on, abrupt or gradual in onset, usually with *pains* in the abdomen and *diarrhœa*, sometimes with nausea and vomiting. The colic becomes violent, constituting actual *tormina*, and the desire to go to stool—*tenesmus*—becomes intense and more or less constant. *Blood* and *mucus* may be absent from the stools at first, but are usually present later on. There is *anæmia*. The disease *affects chiefly the large intestine.*

The *stools* are characteristic. At first there is the discharge of the normal contents of the intestine. There may then be discharged large, dark-brown, thin, offensive stools, in so-called *bilious dysentery*. The stools may contain *scybalæ*, hard fæcal casts of the sacculi of the large intestine. The size of the stools may gradually diminish until there is no passage even upon extreme effort, *dysenteria sicca*. There may be the discharge only of mucus, sometimes with pus, *dysenteria alba;* or blood, *dysenteria rubra*. There may be in the stools inspissated masses of mucus, pus, blood, and débris, resembling flesh in gross appearance, *lotura carnea*. Sometimes the sediment contains particles resembling sago-grains.

Dysentery—complications: Septicæmia, anæmia, peritonitis, perforation, intussusception (rare), hepatic and hepato-pulmonary abscess, pneumonia, pleuritis, pericarditis, tuberculosis, scurvy, malaria, typhoid fever, typhus fever, arthropathies, and paralyses.

Sequelæ: Prolapsus ani, fistula, hemorrhoids; indigestion, irritability of the bowels, constipation; intestinal hemorrhage, ulceration, gangrene, and stricture.

Dysentery—diagnosis: The *catarrhal* form of dysentery is characterized by frequent stools, containing blood and mucus; tormina and tenesmus. This form should be differentiated from luetic and chancroidal ulceration of the rectum, cancer, stricture, the presence of foreign bodies, and intussusception.

The *acute diphtheritic* form of dysentery is marked by a rapid and intense onset, with severe general symptoms resem-

bling those of typhoid fever. The differentiation from typhoid fever may be made by the higher temperature and more severe intestinal symptoms, the stools early containing blood and mucus and later sloughs. The spleen is not enlarged and there are no rose-colored lenticular spots. In the absence of a previous attack of typhoid fever the Widal test will be negative. The diazo-reaction is negative.

The *amœbic* form of dysentery shows the amœba dysenterica in the stools on microscopic examination. The symptoms vary greatly, and not infrequently cases are recognized only after abscess of the liver or lung.

The *chronic* or secondary form of dysentery may not show characteristic symptoms of dysentery. Extensive colitis and ulceration may show only slight diarrhœa. The diagnosis may be made by the use of the rectal speculum and careful physical examination.

Prognosis: The mortality of dysentery is high in the diphtheritic form; low in the catarrhal form. In the individual case the prognosis depends largely on the severity of the symptoms. Complications increase the mortality. Recovery is usually slow.

Prophylaxis: The drinking-water should be pure—at any rate not polluted by excrement. If necessary to drink contaminated water, it should be boiled. The fæcal discharges of dysenteric patients should be destroyed, best by fire.

Dysentery—treatment: Usually recovery occurs spontaneously in about ten days to two weeks. The patient should observe absolute rest in bed. Ipecac is recommended, in small doses, gr. j–iij, repeated every two or three hours; or large doses, gr. xx–xl every four to twelve hours. Opium, internally or in suppositories, is useful in the relief of symptoms. Castor oil, calomel, rhubarb, or the salines, sodium sulphate or magnesium sulphate, may be used to secure purgation. Among the antiseptics, bichloride of mercury, bismuth, creosote, carbolic acid, salol, naphthalin, resorcin, creolin, and tricresol have been used internally. Antisepsis per rectum is more practicable. The colon may be thoroughly irrigated with water, warm or cold, simple or medicated, best injected through the rectal tube with the patient in the knee-elbow

posture. The best results may usually be obtained by rectal irrigation with water containing some astringent, such as alum or nitrate of silver.

Meat is the best diet, especially in the amœbic form of dysentery. Prostration calls for the use of stimulants. Some cases may be cured only by a voyage or a change of climate.

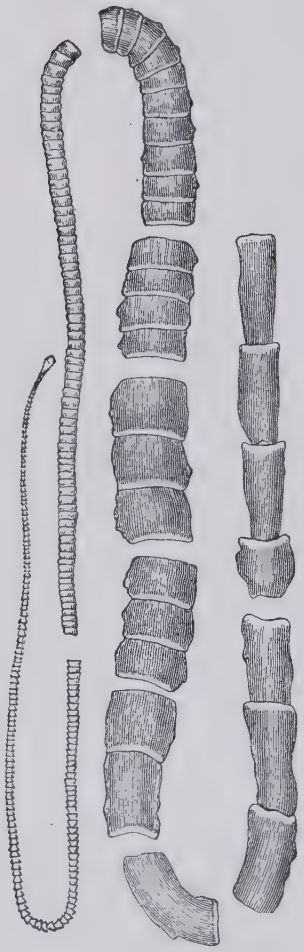
TAPEWORMS; TÆNIÆ; CESTODES.

A **tapeworm** (Fig. 22) consists of a head (scolex), neck, and a body (strobilla) made up of segments (proglottides). The parasites are hermaphroditic. The life-history of a tapeworm (after the *ovum*) is divided into two periods: (*a*) larval or embryonic stage (hydatid, measles, cysticerci), and (*b*) the mature stage. The two stages are usually spent with different hosts. The *ovum* (oncosphæra) after entering the alimentary canal, through contaminated food or drink, finds its way into the tissues of the body, through the bloodvessels or lymphatics, to develop into a hydatid (larval stage). When measles, or cysticerci (larval stage), are ingested, they develop into tapeworms in the intestine.

Etiology: Tapeworms appear in man chiefly as a result of the ingestion of infected food, especially meat that has not been thoroughly cooked. The disease occurs most frequently in early adult life; but age and infancy are not exempt. A case of infection by the tænia armata has been reported in an infant ten weeks old, by Meusiuga (Dock). In that case the infection was carried by contaminated milk.

Tapeworm—symptomatology: The number of symptoms attributed to tapeworm is legion, many of which are doubtless due to helminthophobia. The most important symptom is the passage of segments, either with the stool or, more rarely, by emesis. Among the more common symptoms may be mentioned disturbances of digestion, nausea, vomiting, and eructations. The appetite may be increased, diminished, or capricious. Abdominal symptoms are sometimes complained of, especially after eating certain foods, such as raspberries or strawberries, or foods characterized by a sour or bitter taste, and may be relieved by the ingestion of certain other foods,

Fig. 22.



Sections from *tænia saginata*—natural size (Leuckart).

especially milk. Nervous disturbances are especially common in children. Sometimes there is anæmia, which may be severe. There may be fever.

Peifer believes it probable that the animal parasites contain or excrete *toxic materials*, which act as poisons, especially upon the nervous system and on the formation of blood.

Diagnosis: A positive diagnosis can be made only upon the discovery of segments or eggs. The determination of the variety of tapeworm present in a given case will depend upon an examination of the segments or eggs.

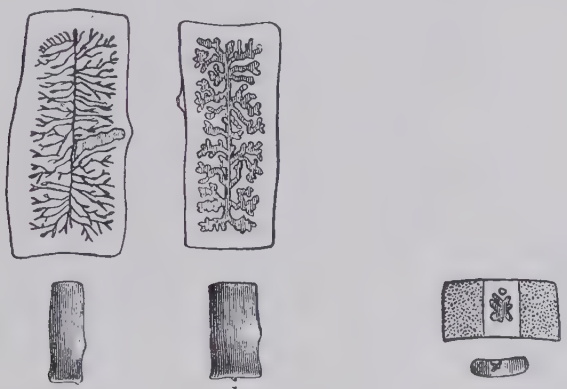
The **prognosis** is usually good under proper treatment. In cases of infection by the *tænia armata* there is danger of cysticercus. Anæmia is especially likely to be severe and possibly fatal in cases of infection by the *bothriocephalus latus*.

Tapeworm — prophylaxis: Animals intended for food should not be fed upon excrement or the offal of slaughter-houses. Meat should be inspected before it is offered for sale, and thoroughly cooked before it is eaten. It is well to remember, in this connection, that the outside of a piece of meat may be burned while the inside remains at a temperature that will not destroy "measles."

Infected individuals should be warned of the danger of

communicating the disease; they should be cleanly. The hands may become contaminated, and should be thoroughly cleaned before they are permitted to come in contact with the

FIG. 23.



a *b* *c*

Proglottides of (*a*) tænia saginata (*b*) t. solium (Leuckart), and (*c*) bothriocephalus latus (Eichhorst); natural size and enlarged three times to show arrangement of uterus.

mouth or with food or cooking-utensils. This advice applies not only to patients, but to physicians as well.

Tapeworm—treatment: For a day or two the individual should fast, or live only upon articles that will afford the

FIG. 24.

a *b* *c*

Eggs of (*a*) t. saginata; (*b*) t. solium; (*c*) bothriocephalus latus; × 300 (Eichhorst).

worm little nourishment and at the same time appease the patient's appetite: black coffee, pickles, cabbage, raspberry jam, wine, and water. A purgative, such as calomel, may be

administered in the evening; and next morning, the patient fasting, a tæniacide may be administered, sometimes best with a cup of black coffee, and the patient instructed to remain in bed.

Among the *remedies* in common use as tæniacides are the ethereal extract or oleoresin of male fern; the rind of the fresh pomegranate root in powder or decoction; or, better, the tannate of pelletierin. Turpentine and pumpkin-seed are used.

If the worm does not pass within an hour, a purgative should be administered; best a Seidlitz powder or a glass of Hunyadi water. If nothing passes within another hour, the patient should receive an enema.

Among other tæniacides are cusso, which should not be given in pregnancy; kamala, used in the form of the powder, 4–8 grammes, or the saturated tincture, 4–12 grammes, given in cinnamon-water; cocoanut, an entire nut, including the milk, to be used within a few hours and followed by free catharsis; black oxide of copper, naphthalin, thymol and myrtol, salicylic acid and salol, and papain.

Upon the expulsion of a tapeworm a search should be made for the head, since if the head remain alive in the alimentary canal the worm may continue to grow. Under such circumstances segments will again appear in the fæces in the course of a few months.

The following **varieties of tapeworm** may be noted:

Tænia Echinococcus.

Synonyms: Dog tapeworm; tænia nana of van Beneden (*not* the tænia nana of v. Siebold).

Description: The tænia echinococcus (Fig. 25) consists of three or four sections, with a total length of 5.5–9.0 mm. (Deffhe). The last section or proglottid is mature, about 2.0 mm. long and 0.6 mm. in breadth, and contains about 500 eggs (30 or 40 hooklets).

Occurrence: The parasite is found especially in the larger varieties of the dog, in the upper half of the small intestine, except ten or fifteen centimetres next the stomach.

Tænia echinococcus—etiology: The parasite exists in man both in the larval stage, as *hydatid cysts*, and in the mature stage, as a "tapeworm." The disease is most frequent in Iceland, and many cases are reported from Australia. In America the disease is not common, and many of the recorded cases were imported. Infection occurs largely through unclean habits. The eggs from the ripe proglottides gain entrance to the alimentary canal of man chiefly through food or drink contaminated by the fæces of the dog.

Fig. 25.

Tænia echinococcus. *a*, natural size; *b*, × 12 diameters (from Whittaker and Ziegler).

Hydatid cysts, the *larval stage* of the echinococcus, may affect almost any part of the body, most frequently the liver, next in frequency the lungs; less often the peritoneum, which is usually affected secondarily; the spleen, kidneys, muscles, bones, central nervous system, and the heart; rarely, if ever, the muscular walls of the stomach, intestine, or bladder, the uterus, Fallopian tubes, and vagina.

Hydatid cyst—symptomatology: The symptoms vary with the organ affected. The growth of the tumor is slow, extending over years. As a rule pain is absent, unless caused by distention of some organ or by pressure on a nerve-trunk. Fever is absent or not characteristic. Often there is urticaria, especially after puncture, sometimes without evidence of traumatism. The hydatid thrill, *frêmissement hydatique*, a peculiar vibration or crepitation, may be felt during percussion.

Diagnosis: Suspicion may be aroused by the presence of a tumor of slow growth and uncertain nature, especially when situated in one of the organs most frequently affected by *echinococcus*, the liver or lungs. A positive diagnosis depends largely upon the *chemical* and *microscopic* examination. Otherwise the differentiation from cancer is sometimes most difficult. The fluid, which may be withdrawn through a fine

needle, before suppuration takes place, is colorless and odorless, usually of a neutral reaction, and with a specific gravity of 1009–1015. Chloride of sodium, which has also been found in other cysts, is present in large amounts, 0.61 per cent. (Munk); phosphates only present a trace. Usually the fluid contains sugar, sometimes uric acid. It is said that there is, as a rule, no albumin; but albumin has been found in hydatid fluid, and is sometimes absent in the fluid obtained from other cysts. Succinic acid is sometimes present, and when found is considered characteristic.

The *microscopic* examination may reveal the *hooklets* or membrane of the hydatid cyst, either of which is positive evidence. The hooklets have a characteristic shape, and the membrane is thick and has a tendency to curl at the edges.

Prognosis: The tumor is of slow growth as a rule. The echinococcus may die, the cyst become sterile, undergo degeneration, possibly absorption, and the patient recover. But such a termination should not be anticipated.

Prophylaxis: Cleanliness should be observed that infection may not be received from dogs. It is not the part of wisdom to receive kisses from dogs after they have performed their customary ablutions.

The infection of the dog may be greatly lessened by not feeding him upon the offal of slaughter-houses, especially the intestines of animals that may contain echinococci.

Hydatid cyst—treatment: The treatment is surgical. If possible, the cyst should be removed entire. Where this is not feasible, aspiration or incision and drainage may result in a cure. Sometimes it is necessary to amputate a member.

Tænia Armata.

Synonyms: Pork tapeworm; tænia solium. The parasite has received the name "pork tapeworm," because its cysticercus (larval) stage is spent most frequently in pork as the *cysticercus cellulosæ* (measly pork).

"Solium," according to Leuckart, is derived from a Syrian word meaning chain.

Description: The tænia armata is 2.0–3.5 m. in length. The

head is spheroidal, 0.6–1.0 mm. in diameter, surrounded by a rostellum and a circle of hooklets, 20–32 in number. The hooklets are of two sizes, 0.11–0.14 and 0.16–0.18 mm. in length, and alternate. The suckers are hemispheroidal, 0.4–0.5 mm. in diameter. The neck is 5–10 mm. long. The proglottides, of which there may be as many as 850, are at first greater in width than in length near the head, the width and length becoming equal about a yard from the head, while in the mature segments the length is about 10–12 mm. and the width 5–6 mm. The eggs are spheroidal, 0.031–0.036 mm. in diameter. The uterus is a straight median canal, from which there are five to seven branches upon either side, given off at right angles.

Occurrence: The parasite in its mature stage occurs only in the small intestine of man.

In the *larval stage*, cysticercus cellulosæ, the parasite occurs most frequently in the hog, as measly pork, and has also been found in the cat, rat, ape, and *man*. The cysticercus cellulosæ is a small elliptical cyst, 6–20 mm. in length and 5–10 mm. in width, in which the inverted head may be seen as a whitish spot. Cysticercus infection in man has been known to result from ripe proglottides entering the stomach from the intestine during the act of vomiting. Cysticerci have been found in the brain, heart, lymphatic glands, liver, bones, tongue, eye, and subcutaneous cellular tissue.

Cysticercus cellulosæ—treatment: Usually the condition is difficult to diagnosticate during life. Cysticerci that do not cause severe symptoms may be let alone. The extract of male fern has been offered as a specific, in 1–3 gramme doses continued for a few days. But in general the treatment is symptomatic. Cysts that can be located may be removed by the surgeon, where such an operation is justified by the symptoms. Aspiration of the cyst is frequently resorted to, sometimes followed by the injection of iodine or alcohol. When considering the feasibility of an operation it should be remembered that the cysticercus may die and become calcified.

The **cysticercus acanthotrias** (Weinland), a variety of the cysticercus cellulosæ, characterized by the arrangement of the

hooklets in three rows, has been reported only once in the muscles and brain.

Tænia Saginata.

Synonyms: Beef tapeworm; unarmed tapeworm. The name, saginata, comes from the word *saginare*, to fatten; but the worm is not always fat.

Description: The parasite (Fig. 22) is 4–11 m. long. The head is cubical, 1.5–2.0 mm. in diameter. There are four suckers, about 0.8 mm. in diameter, usually pigmented. The ripe proglottides are 16–20 mm. long and 4–7 mm. wide. The uterus, situated in the median line, has 20–30 or more branches.

Occurrence: The tænia saginata *occurs* in the human intestine in the mature stage. Ripe segments are prone to escape from the anus and wander about the clothing, more so than the segments of the tænia armata. The eggs are oval, 0.03–0.04 mm. long and 0.02–0.03 mm. broad.

The **cysticercus** of this variety of tapeworm occurs in cattle, hence the name beef tapeworm. The cysticercus may be as large as 9.0 mm. long and 5.5 mm. wide, most frequently situated in the internal pterygoid muscles and tongue, sometimes in the heart, lungs, lymphatic glands, and peritoneum of cattle, and it has been reported found in the brain and eye of man. But it is doubtful whether the cysticercus of the tænia saginata ever occurs in man.

Tænia Cucumerina (Bloch).

Synonyms: Tænia canina; tænia elliptica.

Description: The parasite is 10–40 mm. long and 2–3 mm. wide. The ripe proglottides are 6–10 mm. long, in shape resembling a cucumber-seed. The eggs are spherical, 0.043–0.05 mm. in diameter.

The parasite *occurs* in dogs, cats, and occasionally in human beings, especially in children who fondle dogs and cats.

The *cysticercus* of this tapeworm lives in the dog-louse (trichodectes canis), dog-flea (pulex serraticeps), and human flea (pulex irritans).

Tænia Nana (v. Siebold).

Dwarf tænia: Should not be confounded with the tænia nana of van Beneden (see Echinococcus).

The tænia nana has the distinction of being the *smallest* tapeworm infecting man. Cases have been reported especially from Egypt, Italy, Sicily, and Germany. The parasite is 8–27 mm. long and 0.5–0.7 mm. wide. The head is spherical, 0.25–0.50 mm. in diameter. There are four round suckers and a rostellum containing 24–30 hooklets arranged in a single row. The segments are short, 150–200 in number. The eggs are oval, 0.04–0.05 mm. in diameter. (The eggs do not possess the radiating structure found in the eggs of tænia armata and tænia saginata.)

There may be from 40 to 5000 of these parasites in one individual.

Tænia Diminuta (Rudulphi).

Synonyms: Tænia flavopuncta (Weinland); tænia Minima (Grassi).

Description: Length, 20–60 mm.; width, 3.5 mm. The head is club-shaped and contains four suckers and an unarmed rostellum. The eggs are round, 70–80 μ in diameter. The parasite is common in rats and mice, and has been found in man, especially in children.

The *cysticercus* lives in the caterpillar and in several insects and their larvæ.

Tænia Madagascariensis: Length, 30 cm. Armed. A few cases of infection by this parasite have been reported from the East. The immature stage is supposed to be passed in birds.

Bothriocephalus Latus.

Synonyms: Tænia lata; broad tapeworm; fish tapeworm.

Description: The *largest* tapeworm found in man; it may attain a length of 9.0 m. and a breadth of 10–18 mm. The head is 2–3 mm. long, almond-shaped, with a groove like a sucker on either side. The mature segments have a length of only 5–6 mm. There have been reported as many as 4200

segments in a single parasite. The uterus is described as rosette-shaped, resembling an armorial lily, and is situated in the centre of the segment. The uterus presents some 4–6 convolutions on either side. The male and female genital openings are located behind the uterus, on the flat side in the middle of the proglottis. The eggs are oval, length about 0.07 mm., width 0.045 mm., characterized by a thin membrane and a lid (operculum). The eggs become brown upon exposure to water or air.

Occurrence: The "*measles*" (plerocercoids) of the bothriocephalus latus occur in the muscles and intestines of fish, especially the pike, carp, and salmon. The parasite is not killed by subjecting infected fish to the action of smoke, salt, or freezing. These may develop into mature tapeworms in the dog, cat, and man. Infection from vegetables contaminated with eggs or measles, through irrigation, has been suggested as an explanation for the cases occurring in individuals who do not eat fish.

Infection with the parasite has been found, in this country, only in emigrants.

Bothriocephalus cordatus has been found in dogs, walrus, and seals, in Iceland, and may infect man.

Bothriocephalus leguloides: Found chiefly in eastern Asia.

Bothriocephalus cristatus has been found only occasionally in man.

DISTOMIASIS.

Distomiasis is a disease caused by **trematodes.**

Trematodes, or *flukes*, are flat, sometimes cylindrical parasites, often resembling in shape the tongue or a leaf. The disease is very rare in the United States.

The following are the more important **varieties** of the flukes:

Distoma hepaticum, the *liver fluke*, leberegel (German), douve (French). Found rarely in man, commonly in the liver of the sheep, especially in Arabia. Huber says that "in the Munich abattoirs not a single sheep among many thousands is found free from the liver fluke." The disease is

very rare in the United States. The parasite exists in the immature stage in the snail, from which individuals may become infected through eating contaminated grasses or vegetables (cress).

In the *liver* the parasite produces symptoms mechanically, through obstruction of the biliary passages. Sometimes the parasite is found in the subcutaneous cellular tissue, apparently from infection through the skin.

Distoma magnum (Bassi): Length, 57–73 mm.; width, 24–35 mm. Found most frequently in deer in Italy and in cattle in the United States. The parasite does not occur in man. For a long time it was confounded with the distoma hepaticum.

Distoma lanceolatum (Mehlis): Smaller than the liver fluke. Occurs commonly in the ruminants. A few cases have been reported in man.

Distoma Buskii (Lankester): *distoma cranum* (Busk). Larger than the liver fluke. Infection probably occurs through eating salads, fish, or oysters contaminated by the eggs (Cobbold). The parasite is encountered most frequently in Asia.

Distoma Sibiricum (Winigradoff): Found in man in Tomsk. Length, 13 mm.; width, 3 mm.

Distoma pulmonale (Baelz): 8–10 mm. long and 5–6 mm. wide. Occurs most frequently in the *lungs*.

The *symptoms* are cough, with the expectoration of reddish-brown sputum, resembling the intestines of fish. The sputum contains the eggs in large numbers and sometimes the parasite.

The parasite has been found in the liver-tissue, but *not* in the biliary passages.

Infection of man occurs most frequently in Japan. Cases of infection of the cat and dog have been reported in the United States (Ward).

The *eggs* are 0.1 mm. long and 0.05 mm. wide. They are frequently found in the sputum, sometimes in the brain, liver, omentum, mesentery, mediastinum, and diaphragm.

Distoma spatulatum (Leuckart): *Distoma endemicum* (Baelz). Length, 11–12 mm.; breadth, 2–3 mm. Found commonly in the liver of the cat, sometimes in man, especially in Japan.

The *symptoms* are increased hunger, sensation of pressure

in the epigastrium, and marked enlargement of the liver and spleen; later, sometimes only after several years, there is diarrhœa, sometimes with the passage of blood, ascites, œdema of the lower extremities, and cachexia.

Distoma conjunctum (Cobbold) has been found in the biliary passages of man in Calcutta (McConnell). A similar fluke has been found in the American fox.

Distoma heterophyes (v. Siebold): 2 mm. long and 1 mm. wide. Probably often overlooked on account of its small size (Huber). The skin of the parasite is covered with spine-like scales. The eggs are 0.03 mm. long and 0.17 mm. thick. Cases of infection have been reported from Cairo and Alexandria.

Distomum hæmatobium (Bilharz): *Bilharzia hæmatobia* (Cobbold). Found in various parts of Africa, where infection of man occurs through bathing.

The parasite and eggs are found especially in the *urinary bladder*. The dangers arising from infection have probably been exaggerated. The male parasite, which is the shorter and thicker, receives the slender female into a canal (canalis gynæcophorus) formed by a turning in of the abdominal borders, the anterior extremity of which contains the sexual opening. The eggs are 0.12 mm. long and 0.04 mm. wide, and are characterized by a short spine at the end or to one side.

Amphistomum hominis: Length, 5–8 mm. Has been found in the cecum and colon of man in India.

Amphistoma hawkesii and **amphistoma collinsii** have been found, respectively, in the elephant and horse, in India.

Distomiasis—symptoms: The symptoms depend largely upon the *location* of the parasite, which varies in different species. The parasite may be in the intestine, bile-ducts, gall-bladder, liver, lungs, spleen, bloodvessels, kidneys, ureters, or mesentery. The more common symptoms are diarrhœa, dysentery, hæmaturia, cough, asthma, pyelonephritis, and pyonephritis.

The **diagnosis of distomiasis** depends upon the symptoms; but more especially upon the discovery of the parasite or eggs in the fæces, urine, or sputum.

Distomiasis—treatment: Male fern, in the form of the

ethereal extract or oleoresin, is the remedy in most common use. In the treatment of infection by the distoma hæmatobium (Bilharz), Fourquet recommends the exhibition of male fern, and in bad cases irrigation of the bladder with a solution of bichloride of mercury.

Other remedies are benzene or picric acid (Heller), salicylic acid, salol, naphthalin, and thymol, administered internally.

Rectal injections of sulphuretted hydrogen or carbon dioxide have been recommended in cases of infection by the distoma hæmatobium or distoma pulmonale. It is supposed that absorption of the gas occurs through the hemorrhoidal, vesical, and mesenteric veins, so that the agent comes into direct contact with the parasite.

Flukes may be removed from the bowel by large rectal injections, best given through a colon tube, of solutions of alum, tannin, or creolin, used warm, in conjunction with internal medication.

Further treatment is symptomatic. Anæmia may call for the administration of iron, either alone or in combination with bitter tonics.

NEMATODES (Nematoid (Filiform) Worms).

Ascaris lumbricoides: Round worm; spulwurm (German); lombric (French). One of the most common parasites, occurs in all parts of the world, especially in warm countries, most frequently in children. Males, 15–25 cm. long; females, 20–40 cm. long. The eggs are elliptical, 0.05–0.07 mm. long and 0.04–0.05 mm. wide, and may number 60,000,000 in a single parasite.

The parasite is most frequently found in the small intestine. The usual number of parasites in an infected individual is 2–10; although as many as 5000 are reported to have been passed by an individual within three years.

Round worm—etiology: Infection may be transmitted directly, by means of the hands, through unclean habits; or indirectly, through contaminated food, since freezing or drying, unless long continued, will not kill the eggs.

Nineteen cases of abscess of the liver, due to the ascaris

lumbricoides, have been reported. The parasite probably opened the way for bacterial infection to cause the abscess.

Round worm—symptoms: These may be entirely absent, even in the presence of large numbers of parasites. Itching of the nose is a common symptom, possibly due to a peculiar odoriferous principle in the parasite (Huber). Often there is anæmia caused by malnutrition. Various symptoms may be produced on the part of the alimentary canal: salivation, anorexia, nausea, vomiting (rarely of the parasite); diarrhœa, sometimes with bloody stools; or constipation, obstruction of the bowel sometimes being caused by masses of parasites. Perforation of the intestine has been reported; but many authors believe that the parasite can appear in the peritoneal cavity only in cases of ulcer of the intestine (typhoid fever, tuberculosis). Sometimes symptoms may be caused by the parasite finding its way into the bile-ducts, the vermiform appendix, stomach, œsophagus, glottis, Eustachian tubes, nose, rarely in the lachrymal ducts, urinary bladder, vagina, Fallopian tube, the peritoneal cavity, pleural cavity, or in abscesses. The parasite finds its way into the peritoneal and pleural cavities and the urinary bladder chiefly through fistulæ communicating with the alimentary canal.

More common are *nervous symptoms*, all varieties of which have been ascribed to this parasite.

A positive **diagnosis** can be made only upon finding the parasite or its eggs.

The **prognosis** is good under proper treatment.

Prophylaxis: This calls for cleanliness, which may be difficult to secure at all times, especially in children and the insane. Infected individuals should be instructed regarding the danger of contaminating food. It would be better if the fæces of such persons were disinfected or burned. Infection through the water-supply may be guarded against by boiling or filtering the water.

Round worm—treatment: Santonin is the remedy in common use, in doses of gr. j–iij to an adult; gr. $\frac{1}{4}$–j to a child two years old. The remedy may be given in combination with castor oil or calomel, or in the form of a confection followed by a laxative.

Other remedies are the fluid extract of spigelia, senna, oil of chenopodium, infusions of cusso and kamala, and turpentine.

Ascaris mystax (Zeder): Males, 45–60 mm. long; females, 120–180 mm. long. Upon the sides of the head there are two wing-like appendages. The eggs are spherical, 0.068–0.072 mm. in diameter. The parasite is commonly found in the dog and cat, and has been found in man.

Ascaris maritima (R. Leuckart): A female 43 mm. in length, not yet mature, vomited by a child in Greenland, has been reported by Leuckart.

Oxyuris vermicularis, Ascaris vermicularis; pin-worm; seat-worm; thread-worm. Males, 3–5 mm. long, posterior extremity blunt and curled up; females, 10 mm. long and 0.6 mm. wide. Eggs, 0.05 mm. long and 0.02 mm. wide. The parasite may contain from ten to twelve thousand eggs.

Fig. 26.

Oxyuris vermicularis. *a*, male; *b*, female; natural size (Eichhorst).

Fig. 27.

Eggs of oxyuris vermicularis. × 275 (after Eichhorst).

These are usually passed after the parasite leaves its host. V. Jaksch claims to have found the eggs in the fæces in almost all cases; but other observers (O. Leichtenstern, Lutz, Huber) have failed to find them. The parasite is found in the small intestine. After impregnation the female passes into the large intestine. Eggs may sometimes be found in the lower part of the colon. As a rule, however, the eggs are not de-

posited until the female has passed out of the alimentary canal.

Etiology: Infection follows ingestion of the eggs of the parasite. Food may be contaminated. More frequent is direct infection through unclean habits. The eggs are introduced into the mouth from the hand, which has become contaminated, as a rule, through the efforts to relieve the itching caused by the parasite.

Pin-worm—symptoms: The most prominent symptom is the burning sensation, the itching of the anus, caused by the sharp end of the female. The symptom may not appear until the individual has retired at night. Numerous symptoms on the part of the nervous system, including convulsions, may be caused, in a susceptible individual, by the irritation of the parasite.

The **diagnosis** is usually made by an examination of the anus, which will reveal the parasite. Should the parasite not be found upon examination, it may be seen after the injection of cold water.

Prognosis is good.

Prophylaxis demands cleanliness. As stated, infection is usually carried to the mouth by the unclean hand that has been used to relieve the itching of the anus caused by the parasite.

Treatment: Usually an enema of water, plain or containing salt, alum, or creolin, will suffice to remove the parasite. Parasites in the small intestine may be removed by the administration of santonin and a laxative.

Schmitz reports success with *naphthalin*. After the administration of a purgative, naphthalin in powder (0.15 for a child one and a half years old to 0.4 at twelve years), given four times a day for two days, and repeated after eight days, is effectual.

Trichocephaliasis: Infection by the trichocephalus hominis (Schrank); trichocephalus dispar (Rudolphi); whip-worm. Named "trichocephalus" from the resemblance of the head to a hair. The tail end is much larger than the head. The parasite is 40–50 mm. long and 1.0 mm. wide. The male is shorter than the female, and has a body that is rolled up into

a spiral. The eggs, 0.05 mm. in diameter, are lemon-shaped, brown colored, with thick shells and projecting clear extremities. There may be as many as 58,000 eggs in a single parasite (Leuckart). The eggs resist drying and changes in temperature. Infection occurs chiefly through the ingestion of contaminated food or water. The number of parasites in an individual usually varies from 3 to 10, although more than a thousand have been reported (Rudolphi). The parasites are usually found in the cæcum and appendix vermiformis, large intestine, and sometimes in the lower part of the small intestine.

Whip-worm—symptoms: Frequently there are no symptoms. The intestinal irritation may cause diarrhœa, sometimes bloody stools, nausea, vomiting, and emaciation, with anæmia and loss of strength. Nervous symptoms may be more or less marked.

The **diagnosis** is made by the discovery of the eggs or the parasite; but the parasite does not often appear in the stools.

The **prognosis** is good as a rule, although sometimes the parasite is not easily dislodged.

Prophylaxis calls for cleanliness, that infection may not occur directly or food or drink become contaminated. A contaminated water supply should be remedied, or the water filtered or sterilized by boiling before it is used.

Treatment: This is the same as that outlined for oxyuris vermicularis, except that it is important that the enemata should be sufficiently large to reach the cæcum. A combination of male fern internally and rectal enemata is sometimes effectual where other means fail.

Ankylostomiasis; Dochmiasis; Egyptian chlorosis; *tropical chlorosis;* brickmakers' anæmia; miners' cachexia; caused by the *ankylostomum duodenale* (Dubini), strongylus quadridentatus (v. Siebold). Female, 6–18 mm. long and 1.0 mm. wide; male, 6–11.5 mm. long and 0.5 mm. in diameter. There are more females than males. During copulation, which lasts for several days, the union between the parasites is so firm that attempts at forcible separation may result in tearing the body of the male. The eggs are 0.66 mm. long and 0.03 mm. in

diameter. The number of eggs passed is very great; in an evacuation of a pint Leichtenstern estimated the number of eggs at four millions. After escaping from the intestinal canal the eggs become encysted. When these are ingested they lose their outside covering through digestion in the stomach, and the embryos become attached in the intestine, usually in the jejunum, more rarely in the duodenum, exceptionally in the pylorus, stomach, or ileum.

Tropical chlorosis—etiology: The disease occurs in those brought into intimate contact with soil contaminated by fæces (brickyards, mines, tunnels), largely through infection of the food by dirty hands, or the use of water containing eggs of the parasite. Lutz has reported numerous cases occurring in children in Brazil. The number of parasites present in an individual is rarely less than a hundred, and may be over three thousand (Grassi).

Symptomatology: The parasite extracts blood from the intestinal wall, living upon the serum of the blood. Consequently there are anæmia, sometimes the appearance of blood in the stools and evidences of intestinal irritation; and pain and tenderness in the region of the jejunum. There may be fever, salivation, disturbances (increase or decrease) of the appetite, nausea and vomiting, sometimes tympanites. A very common symptom is œdema, beginning in the eyelids and becoming general. With weakness of the heart cyanosis may appear. The number of red blood-corpuscles may be decreased to less than a million to the cubic millimetre, with a corresponding decrease in the hæmoglobin. With this there may or may not be leucocytosis. Men may show impotence; women, amenorrhœa.

The **diagnosis** depends upon the discovery of the eggs, as a rule, which are discharged in enormous numbers with the fæces. Spontaneous discharge of the parasite rarely occurs.

The **prognosis** depends upon the condition of the patient at the time treatment is begun. Even severe cases may recover after removal of the parasites.

Prophylaxis: Food should be thoroughly cooked; the water pure, boiled; and, above all, the hands should be washed

before eating. Infected workmen should be excluded from non-infected localities.

Tropical chlorosis—treatment: The preparatory treatment is the same as that for tapeworm in general. Thymol is highly recommended: four drachms divided into two powders, taken two hours apart, followed in two hours by a dose of castor oil or calomel. Male fern has been largely used. After removal of the parasite tonics, especially iron, should be given.

Eustrongylus gigas is found in various carnivorous animals, sometimes in herbivora, and has been reported in man. The male is 25–35 cm. long; the female, 25–100 cm. long and 5–12 mm. in diameter. The color is reddish. The parasite occurs especially in the kidney, sometimes in the bladder, and is recognized by finding the eggs in the urine. The eggs are 66 μ long and 43 μ in diameter, brown colored. On the upper surface are little depressions with a surrounding elevated border. The poles are flat.

Dracontiasis, Guinea-worm disease, caused by the dracunculus Persarum (Kämpfer), filaria medinensis (Gmelin), Guinea-worm. The parasite is 50–100 cm. long and about 1.7 mm. in diameter. Only the female was known until Charles found a pair in the act of copulation, in the mesentery. The male was about one-third the length of the female (Huber). It would seem that the vagina is in the centre of the body of the female and after copulation becomes occluded. It is probable that both male and female find their way from the intestine to the mesentery, death of the male then occurring after copulation, the female entering the connective tissue and travelling toward the feet. In the great majority of cases the female parasite finds its way to the surface of the foot about the dorsum or malleoli, rarely in the legs or thighs, very rarely about the thorax. The embryos are discharged by rupture of the parent worm. The eggs then find their way into water, where they enter the body of a small fish, and grow to be about 2.0 mm. long, when they are taken into the body of man through the medium of contaminated

drinking-water. Some investigators believe that infection of man may occur directly through the skin from contact with contaminated water.

Guinea-worms—symptomatology: Symptoms are absent until the parasite reaches the skin, when there are localized pain, redness, and swelling, and later perforation of the skin and the discharge of embryos.

The **diagnosis** rests upon the discovery of the embryos or the parasite.

The **prognosis** as a rule is good.

Treatment: The parasite must be carefully extracted when it appears at the surface. This should be done very gradually by winding the parasite upon a piece of wood, a little each day for a number of days, to avoid rupture of the parasite and the discharge of embryos. Good results have been claimed for the injection of bichloride of mercury, 1 : 1000, into the parasite or its burrow. Where it is impracticable to open the burrow, carbolic acid, 1 : 15, may be applied externally by means of compresses.

Filariasis, a disease caused by the *filaria sanguinis hominis* (Lewis); filaria Wuchereri (d'Silva and Lima); filaria Bancrofti (Cobbold). The chief varieties of this parasite are: 1. Filaria sanguinis hominis *nocturna*—male, 83 mm. long and 0.4 mm. in diameter; female, 155 mm. long; eggs, 38 x 14 μ. 2. Filaria sanguinis hominis *diurna*, or *majora*, distinguished by being found in the blood only during the daytime. 3. Filaria sanguinis hominis *minora*, or *perstans*, only the embryos of which are known, 0.2 mm. long.

Filariasis is a disease of the tropics, occurring most frequently in Brazil, the West Indies, and also in Mexico, South America, the South Sea Islands, Japan, Australia, China, India, Egypt, and Spain. The disease is seldom seen in the southern part of the United States.

Symptoms: These may be entirely absent. There may be leucocytosis, chyluria, or hæmatochyluria, and discharge of the parasite and eggs with the urine. The parasite is believed to cause at least some cases of elephantiasis Arabum, lymph-scrotum, lymph-vulva, chylous ascites, chylous diar-

rhœa, possibly the sleeping-sickness of Africa; and a case of filarial hæmoptysis has been reported (Yamane) in which filariæ were found in the expectorated blood. Some observers believe that the mosquito is the intermediate host.

The **diagnosis** depends upon the discovery of the filaria in the blood, which should be examined both day and night. As a rule the parasite appears in the blood only at night; but in some cases it has been found present only during the day, especially in individuals who sleep during the day. The examination of the blood should be made with a low-power lens.

Prognosis: The mortality is not high. The loss of chyle may lead to death from marasmus.

Filariasis—treatment: This is largely symptomatic. Cures have been reported after the use of gallic acid, picro-nitrate of potassium, iodide of potassium, benzoic acid, quinine, and methylene-blue. A cure may or may not follow removal of the parasite. The use of tonics, especially iron, is usually called for.

Trichiniasis.

Trichiniasis, trichinosis, is due to the *trichina spiralis* (Richard Owen).

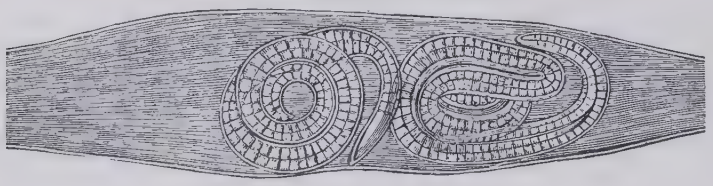
The *infection* occurs in man through the use of trichinous pork, and has been produced experimentally in dogs and cats. Ingested muscle trichinæ are liberated by digestion in the stomach, and in the intestine become sexually mature within about three days and full grown within a week. The males are 0.8–1.5 mm. long; the females, 1–4 mm. long. The genital opening of the male is at the extremity; that of the female is in the neck. Soon after copulation the male dies; the female continues to live, as a rule, about a month longer. The embryos, 0.08–0.12 mm. in length, leave the shell before parturition and are born directly into the intestinal follicles, in which the female trichinæ may sometimes be found inserted.

The *embryos* migrate to the *muscles*, probably along the lymph-channels; according to some authors, along connective-tissue routes; and they have been found in the blood. In the muscles they destroy some of the contractile substance, and after reaching a length of about 0.06 mm. they roll up into a

spiral (Fig. 28), become encapsulated, and continue to grow until they reach a length of about 1.0 mm. In this condition they may remain alive for a number of years—seven to ten, even thirty years in reported cases.

Trichinosis—symptomatology: Soon after the ingestion of infected meat there may be anorexia, nausea, vomiting, abdominal pain, and diarrhœa, sometimes with bloody stools. Usually there are no symptoms until about the end of the first week, when there may be anorexia, malaise, flushes of

FIG. 28.



Muscle-fibre with trichinæ (Heller).

heat, and chilly sensations, sometimes with sweating, thirst, and diarrhœa. An early symptom is muscular weariness.

With the invasion of the *muscles* by the parasite, about the second week, the affected muscles become tender, painful, stiff, swollen, and hard. Usually there is fever, which may reach 104°–106° F., usually highest from the ninth to the eleventh day (Mosler). Fever may be absent even when there is considerable affection of the muscles.

Œdema, appearing first in the eyelids, usually is present after the first week, and lasts as a rule two to five days in the eyelids, longer in the extremities. There may be œdema of the scrotum or prepuce, sometimes ascites.

On the part of the *respiratory system* there may be bronchial catarrh, pneumonia, especially hypostatic pneumonia. Affection of the respiratory muscles may lead to dyspnœa; affection of the laryngeal muscles may cause hoarseness and aphonia. Sometimes there is œdema of the glottis.

On the part of the *nervous system* insomnia is a prominent symptom. There are often headache, and sometimes loss of the tendon-reflexes.

Examination of the *blood* reveals a marked increase of the eosinophile cells in association with *leucocytosis.*

Diagnosis: The symptoms present may lead to the suspicion of trichiniasis. The differential diagnosis concerns especially poisoning by meat and sausage, and acute polymyositis (Unverricht). A positive diagnosis can be made upon the discovery of the parasite in the stools, especially after purgation, or in the muscles of the patient, or in a remnant of meat from which the patient has been eating. There are leucocytosis and a large increase of eosinophile cells.

Prognosis: The mortality varies from 2 to 30 per cent., probably as a rule about 20 to 25 per cent.

Prophylaxis: This in man may be secured by thoroughly cooking pork or abstaining from its use altogether. Infected animals should not be used as food. Hogs should not be fed on the offal of slaughter-houses, and it has been suggested also that they should be protected from rats, which some believe capable of communicating the disease to hogs.

Trichinosis—treatment: Cases seen soon after the ingestion of infected meat may be cured by *lavage of the stomach* and the administration of a *purgative.* Even later in the course of the disease *purgation* may be resorted to in order that fewer eggs may be deposited within the body.

Following the purgation remedies may be given to kill the parasite in the intestine. For this purpose the remedies in common use are keratin-coated pills of the sulphocarbolate of sodium, two grammes every four hours, and salicylic acid, one gramme three times a day. Diarrhœa, which is not too severe, should be encouraged, especially as long as the parasite may appear in the stools. The treatment of muscle trichina is purely symptomatic.

CHAPTER II.

DISEASES OF THE ORGANS OF DIGESTION.

DISEASES OF THE MOUTH.

GENERAL CONSIDERATIONS.

Diseases of the mouth present symptoms which vary largely according to the part of the mouth affected. The buccal secretion is furnished by the salivary and mucous glands, and gives normally an alkaline or a neutral reaction. An acid reaction may be caused by acid fermentation. Decomposition of food may cause the breath to become fœtid. A bad odor from the mouth is known as *stomatodysodia*. In infants, especially during the first three or four months, the buccal secretion is slight, and the mouth is rendered liable to infection by frequent insults. In adults the secretion is diminished, especially in the infectious diseases and whenever there is fever. In such cases a coating forms on the tongue. Mouth-breathing, through drying the buccal secretion, may cause apparent diminution of the secretion.

Hypersecretion (salivation) is found in many diseases, especially in local inflammatory diseases of the mouth. A condition simulating salivation may be caused by obstruction of nasal respiration or motor disturbances preventing the closure of the lips, or by inability to swallow.

General symptoms: *Diseases of the vestibulum and cheek pouches* are characterized by difficult or painful mastication. *Diseases of the tongue* show not only difficult or painful mastication, but also difficulty in swallowing solid food. *Diseases of the soft palate, arches of the palate,* and *isthmus of the fauces* offer difficulty in swallowing. Motor disturbances of the velum palati, and perforations and defects of the hard or soft palate, permit the entrance of food into the naso-pharynx and

its expulsion through the nose. *Motor disturbances of the epiglottis and root of the tongue* permit the entrance of food into the larynx and trachea. *Cleft palate and defects of the palate*, presenting a communication between the buccal and nasal cavities, and *diseases of the velum palati*, interfering with shutting off the naso-pharynx, give rise to a *nasal tone* of voice. When the normal communication between the buccal and nasal cavities is partially or entirely cut off (*tumors*) the *nasal tones are absent*. Disease of the *tongue* affects especially the enunciation of the *explosives and sibilants*. Diseases of the *vestibule, the cheek-pouches*, and especially of the *lips*, affect the enunciation of the *vowels and labial consonants*.

Effects of drugs: The *salivary secretion* may be *diminished by atropine* or *increased by pilocarpin, muscarin, eserin, etc. Iodine and bromine, or their salts*, increase the salivary secretion and cause swelling of the mucous membrane, especially about the gums. A yellowish-white coating appears at the edges of the gums and on the teeth, and the breath has a peculiar odor. *Phosphorus-poisoning* causes similar symptoms and early involvement of the alveolar processes, periostitis, and necrosis of the maxillary bones. *Mercurial poisoning* causes stomatitis with salivation (see Stomatitis Ulcerosa). Chronic *lead-poisoning* causes swelling of the mucous membrane, especially at the edges of the gums, with a deposition of lead, constituting the "lead-line." *Poisoning by silver* (argyria) causes staining of the mucous membrane of the mouth and of the skin of the body, the stain appearing in blackish-brown punctate spots.

Eruptions: *Herpes* frequently involves the mucous membrane of the mouth. Many diseases, especially the *acute infections*, show an eruption in the mouth.

Injuries of the mouth may be caused by the teeth or by external violence, burns, or caustics.

STOMATITIS CATARRHALIS (Acute, Simple, or Erythematous Stomatitis).

Etiology: Improper care of the mouth, especially of the teeth, and irritants of all sorts, act as causes. *Infants* are

especially liable to the disease, because the saliva is small in amount and comparatively inactive, and also because of the habit of putting things into the mouth. In *adults* the use of alcoholic beverages is a prominent etiological factor.

Symptomatology: The *mucous membrane is swollen, dry,* and *inflamed;* later, the salivary secretion becomes increased and there is *pain.* There may be actual salivation and spongy gums, with falling out of the teeth. In severe cases the tongue may become coated and swollen. The sense of taste is diminished or lost. There may be fever, and nutrition may be impaired, especially in infants, who are not able to nurse because of the soreness of the mouth.

Treatment: The mouth should be kept clean and carious teeth should be properly treated. Mouth-washes containing antiseptics, such as thymol (1 : 2000), alum (1 : 200 to 1 : 500), permanganate of potassium (1 : 2000 to 1 : 5000), chlorate of potassium (1 : 25 to 1 : 100), are useful. The local application of iodoform has been highly recommended for ulcerations.

STOMATITIS ULCEROSA.

Etiology: Stomatitis ulcèrosa occurs most frequently in children, especially from five to ten years of age. Sometimes the disease becomes endemic in children's hospitals or wherever individuals are crowded together. It has been suggested that such endemics may be outbreaks of foot-and mouth disease, or of some disease showing complications on the part of the mouth. Besides the causes enumerated in the etiology of stomatitis catarrhalis, the following deserve special mention as causes of stomatitis ulcerosa: the accumulation of tartar on the teeth, syphilis, rickets, diabetes mellitus, tuberculosis, and mercurial poisoning.

Miller found a motile bacillus and spirochæte, always associated, in carious teeth; and the same organisms were found by Bernheim in thirty cases of ulcerative stomatitis in the deposit on recent ulcers.

Symptomatology: The symptoms of stomatitis catarrhalis are usually present in aggravated degree, and in addition there is *ulceration,* which always begins on the gums, usually

about the lower incisor and canine teeth. The ulcers extend to involve the cheeks and lips, frequently the tongue and floor of the mouth. There is a *peculiar foul odor*, with *pain and salivation*. Difficulty in speaking, chewing, and swallowing is marked.

Treatment: The cause should be removed. The mouth must be kept clean. Early in the disease antiseptic mouth-washes may be advantageously used, such as solutions of potassium chlorate (1:25), borax (1:30), potassium permanganate (1:2000 to 1:5000), and tincture of myrrh or of rhatany (gtt. xx–xxv in a wineglassful of water). Early, before sloughing, the borders of the gums may be touched with the stick of nitrate of silver. Later the local application of iodoform, or the insertion of iodoform-gauze between affected contiguous portions of the tongue, lips, and cheeks, is recommended, with the internal administration of potassium chlorate, 2.0–5.0 (gr. 30–75) daily. Periosteal abscesses should be evacuated and sequestra removed. Nutrition must be maintained, if necessary, by feeding through a tube.

STOMATITIS APHTHOSA (Aphtha; Stomatitis Herpetica; Canker).

Definition: An acute infectious disease of the mucous membrane of the mouth characterized by the early appearance in the epithelium of small circumscribed white fibrinous spots surrounded by a bright-red line.

Etiology: Stomatitis aphthosa occurs especially in children during the first dentition. But dentition is not a cause of aphtha. The disease is believed by some to be a form of herpes occurring in the mouth. Friedreich, by the injection of the toxins of the streptococcus and bacillus prodigiosus, produced herpes of the face in seven cases and aphtha in two cases. Fränkel found the staphylococcus citreus and flavus (see Foot-and-mouth Disease).

Symptomatology: The white or yellowish fibrinous patches, sharply circumscribed, round, or oval, appear early, each surrounded by a slightly elevated red border. These cause a burning sensation and actual pain, and when the patches are

numerous or extensive there may be marked interference with mastication, deglutition, and speech.

Diagnosis: *Stomatitis aphthosa* should be differentiated especially from foot-and-mouth disease, in which there are more severe stomatitis and greater depression. The diseases are believed by some investigators to be closely related.

The **prognosis** is good.

Prophylaxis: Most cases may be prevented by cleanliness of breasts, or nursing-bottles, especially the nipples. The mouth should be kept clean. This should include attention to the teeth, the temporary as well as the permanent set, and the proper treatment of carious teeth.

Treatment: The mouth may be washed with a solution of borax in water, or boric acid in glycerin and water. Almost any mild antiseptic solution may be used. Cleanliness is of most value.

BEDNAR'S APHTHA (Bednar's Plaques).

Characteristics: Circumscribed thinning of the epithelium with sharply defined yellow spots on the posterior lateral border of the hard palate. The *etiology* is not clear. The thinning of the mucous membrane has been held to be due to the general desquamation following birth, to pressure of the tongue in nursing, and to *injury of the mucous membrane in cleansing the mouth*. A similar thinning of the mucous membrane has been described in the stillborn. *Cure* should be exercised to avoid trauma in cleansing the mouths of infants. *Recovery* from Bednar's aphtha occurs spontaneously in the course of a few days.

STOMATITIS GANGRÆNOSA (Noma; Cancrum Oris).

The **gangrenous process** begins on the cheek, near Steno's duct, and rapidly spreads.

Etiology: Various micro-organisms have been isolated from the necrotic tissue. Bacilli have been described especially by Schimmelbusch (1889) and Foote (1893); but the etiological relation of none of these has been definitely established.

Since the immediate cause is not known, Forchheimer holds that the *predisposing cause* is the more important.

The disease prefers children—those that are anæmic and not well nourished. Adults may be attacked. Noma has followed measles in about half the cases of noma reported, and in other instances has occurred after scarlatina, typhoid fever, pneumonia, and ulcerative stomatitis (ptyalism).

Noma—symptomatology: The disease comes on with the usual symptoms of stomatitis. A blue vesicle appears on the mucous membrane opposite the first or second molar tooth, near Steno's duct, later becoming darker in color. There is the characteristic odor of gangrene. The cheek shows extensive swelling, with great hardness from infiltration and at the same time pallor. The vesicle breaks down in a gangrenous process, which rapidly extends to perforate the cheek and cause great destruction of tissue. Finally a line of demarcation may form, the necrosed tissue be cast off, and recovery take place, often with great deformity. More frequently death ensues from exhaustion, sepsis, or malnutrition. The disease usually lasts about one or two weeks. Rarely the process may begin in the anterior sublingual region, still more rarely upon the external genitals, the anus, or the auricle.

Diagnosis: Usually easy. At first there might be some hesitation in the differentiation between the early stage of stomatitis gangrænosa and stomatitis ulcerosa.

Prognosis: As a rule bad. The mortality has been given at 70 per cent. Cases that recover usually show great deformity.

Noma—treatment: Early extirpation with the knife or cautery has been recommended; but often gangrene recurs in the wound. Sepsis and malnutrition call for special attention.

STOMATITIS MYCOTICA (Thrush; Stomatitis Parasitica; Soor (German); Muguet (French)).

Thrush was the first disease proven to be due to the action of vegetable parasites.

The specific *cause* of the disease is the thrush-fungus, called by its discoverer the *Oidium albicans*, which has since been placed by some among the highest of the fungi, by

others among the moulds, while still others believe it to belong to neither class.

Stomatitis mycotica *occurs* especially among children, sometimes among invalids. Lack of cleanliness, especially with regard to the mouth, is a predisposing cause. Thrush seems to show a preference for individuals who live upon a milk-diet.

Thrush—symptoms: The disease appears first as small white points or patches upon an otherwise apparently healthy mucous membrane. These patches extend rapidly. The membrane may be removed readily. There is pain, which may interfere with the child's nursing and thus cause malnutrition.

Prognosis: Especially in individuals who are ill, the nutrition may be still further interfered with by the occurrence of thrush, which thus sometimes becomes a forerunner of death; but as a rule the prognosis is good.

Prophylaxis: Thrush may be prevented by proper care of the mouth. The mouth should be kept clean.

Thrush—treatment: Food or medicine containing sugar must be avoided. The mouth should be thoroughly cleaned, probably best with a solution of borax in water or glycerin; a mild solution of potassium permanganate or potassium chlorate; or of methylene-blue; or an astringent mouth-wash (see Stomatitis Ulcerosa).

The **affections of the mouth** by gonorrhœa, syphilis, tuberculosis, glanders, leprosy, and actinomycosis, are treated of, respectively, among the *Infections*.

Among the **animal parasites** found in the mouth may be mentioned the larvæ of eggs of flies; the oxyuris vermicularis; cysticerci; echinococci; filaria Medinensis, and trichina.

DISEASES OF THE TONGUE.

The **tongue** shows great resistance to disease, notwithstanding its constant exposure to "insult."

ACUTE GLOSSITIS.

Acute glossitis is *caused* most frequently by traumatism, burns, and foot-and-mouth disease. *Abscesses* of the tongue may occur as complications of stomatitis or from wounds, occasionally in some of the infections, especially typhoid fever, erysipelas, smallpox, and anthrax. There are pain, salivation, and enlargement of the tongue. The presence of pus is rarely revealed by fluctuation. There may be fever, headache, and loss of appetite.

Diagnosis calls for differentiation from hypertrophy or lymphangioma and syphilitic gummata.

The **treatment** is surgical. The part may be scarified or incised, and idoform-gauze applied.

CHRONIC GLOSSITIS.

A **chronic enlargement** of the tongue may result from repeated attacks of acute glossitis or from myxœdema or cretinism. The *treatment* should address the underlying cause or disease.

MACROGLOSSIA.

Macroglossia, or enlargement of the tongue, is usually congenital; sometimes due to lymphangioma or muscular hyperplasia; rarely to acromegalia.

The **treatment** is surgical. The tongue may be reduced in size by the removal of a wedge-shaped piece.

LINGUA GEOGRAPHICA (Geographical Tongue; Mapped Tongue).

Appearance: The tongue shows, in this condition, smooth red patches denuded of epithelium and bounded by white margins, which show frequent variations in outline, sometimes disappearing entirely to reappear later. Usually the patient complains only of the accompanying stomatitis.

Diagnosis: The geographical tongue should be differentiated from a chronic superficial glossitis, which causes more pain and runs a more chronic course; and from the mucous patches

of syphilis, which shows less variation in outline and longer duration.

Treatment: Medication is unnecessary, further than address to any accompanying stomatitis.

ANGINA.

Definition: Affection of the fauces, pharynx, and throat, especially of the fauces and tonsils, attended with pain, dysphagia, and dyspnœa.

Forms: *Angina* involving the *fauces* may be catarrhal, rheumatic, or herpetic; involving the *tonsils:* lacunar, follicular, or phlegmonous. Angina of the whole throat may be septic, gangrenous, erysipelatous, etc.

The "sore throat" of scarlet fever, diphtheria, syphilis, and tuberculosis has received separate consideration.

DISEASES OF THE TONSILS.

ACUTE FOLLICULAR TONSILLITIS (Acute Catarrhal Angina; Acute Amygdalitis; Croupous Tonsillitis; Lacunar Tonsillitis).

Etiology: Numerous bacteria, especially the micro-organisms of pus and the micrococcus pneumoniæ crouposæ, have been found in the crypts of the tonsil, but not invading the tissue. Grey, Edwards, and Severn traced an epidemic of follicular tonsillitis through the milk-supply to a single animal by bacteriological examinations, which revealed in the throat of an affected case and in the milk from the diseased animal the staphylococcus aureus and albus and the short form of the streptococcus pyogenes. Goodale has suggested that possibly the disease may be due to the absorption through the mucous membrane of irritating toxins formed in the crypts as in a test-tube. The cases in which the micrococcus pneumoniæ crouposæ is found in pure culture usually show a sudden onset and a termination by crisis, resembling the picture seen in croupous pneumonia (Vent, Jaccoud). The pseudomembranous angina, appearing about the fourth day of scarlet fever, usually shows the streptococcus upon bacteriologic

examination. Cases of pseudodiphtheria also present the streptococcus, which is often very virulent.

The disease prefers young adults, and occurs especially in spring and autumn, sometimes in epidemic form.

Acute tonsillitis—symptomatology: Early the patient complains of tickling or burning in the throat. There is dysphagia, sometimes dysphonia. There may be salivation. The tonsils are inflamed, and on them are found yellowish-white spots, which may not be easily removed. The spots or patches of exudate are separated by apparently healthy tissue. The patches are usually confined to the tonsils and when removed do not leave a denuded surface.

The general symptoms may be scarcely noticeable or quite severe. Usually the disease begins with chilly sensations; sometimes with an actual chill. As a rule there are lassitude and malaise, sometimes severe prostration. There are headache and anorexia, sometimes nausea and vomiting. The temperature may reach 102°–105° F. The pulse and respiration are rapid. Examination of the blood shows marked leucocytosis. The spleen may be enlarged. The urine varies with the fever, and may contain albumin and casts.

Acute tonsillitis—diagnosis: This concerns chiefly the differentiation from diphtheria. It is better to make a bacteriological examination for the diphtheria bacillus before excluding diphtheria. In acute follicular tonsillitis there will usually be found streptococci or the micrococcus pneumoniæ crouposæ.

The **prognosis** is usually good. The rare fulminating septicæmic forms may result in death. Pseudomembranous angina, especially that occurring in scarlet fever, may extend to the nose, ear, or larynx. Endocarditis and nephritis have been reported.

Acute tonsillitis—treatment: This is symptomatic. For the toxic symptoms phenacetin affords considerable relief, and may be given in gr. x doses, combined with caffein and sodium bicarbonate, āā gr. j, repeated two or three times a day for a day or two. One of the best remedies is salicylate of sodium. Local treatment, especially cleansing the tonsil with antiseptic solutions, is of value.

SUPPURATIVE TONSILLITIS.

See Quinsy.

HYPERTROPHY OF THE TONSILS (Chronic Tonsillitis).

Hypertrophy of the tonsils occurs especially in children, and has been found in the new-born.

Symptomatology: The patients frequently have repeated attacks of acute tonsillitis and "catch cold" on slight exposure. Usually there is mouth-breathing; sometimes interference with breathing, rarely with swallowing. Occasionally hypertrophy of the tonsil causes torticollis.

Diagnosis is easy. Distention of the tonsil from concretions or cheesy masses may be eliminated by careful examination with a probe. Malignant disease usually occurs later in life.

Prognosis: As a rule hypertrophy of the tonsil disappears after puberty. In the meantime cases that are not properly treated may suffer permanent injury. Infection occurs more readily and diphtheria is more severe.

Chronic tonsillitis—treatment: The tonsil should be removed, possibly best with a tonsillotomé or in adults with a cold-wire snare. Hemorrhage is usually of short duration in infants; but in adults may be severe and require torsion, pressure, or possibly even ligation of the common carotid. Usually such hemorrhage may be avoided by the use of the cold-wire snare.

DISEASES OF THE PHARYNX.

ACUTE PHARYNGITIS.

Etiology: Acute pharyngitis is frequently ascribed to "taking cold." Probably the most frequent causes are impure air, loss of sleep, and fatigue. The disease is frequently observed in connection with diseases of the stomach. Obstruction of the nose is an important etiological factor in many cases.

Symptomatology: There are tickling in the throat, a dry cough, and a persistent desire to swallow. Inspection reveals

the mucous membrane of the pharynx reddened, possibly streaked with secretion from the naso-pharynx. Dysphagia would indicate involvement of the tonsils. The constitutional symptoms are not so marked as in tonsillitis. The attacks usually last from three to six days.

In **diagnosis** the differentiation from diphtheria is most important. All doubtful cases should be examined bacteriologically.

Acute pharyngitis—treatment: Symptomatic. Inhalations of steam, simple or medicated, and hot gargles give most relief. Any underlying disease on the part of the stomach or nose should be remedied. Fever may call for quinine or phenacetin.

CHRONIC FOLLICULAR PHARYNGITIS.

Chronic follicular pharyngitis usually begins in childhood, when the disease is frequently overshadowed by enlargement of the tonsils. Excessive use of the voice probably plays a rôle in *etiology*.

Symptomatology: Often there are dull pain and tickling in the throat, hoarseness, dry cough, and hawking. Examination reveals the hypertrophied pharyngeal lymph-follicles, bright red in color, and sometimes redness of the entire pharynx. There will not be increased secretion, nor will the uvula be elongated, unless there is a complicating naso-pharyngeal catarrh.

Prognosis: The disease is chronic but not serious.

Treatment: Excessive use of the voice should be avoided, and the patient should be placed under good hygienic surroundings. Further than this the treatment is surgical. The hypertrophied follicles should be removed, best by the cautery, actual or galvanic.

RETROPHARYNGEAL ABSCESS.

Etiology: Most cases occur in infancy. Traumatism has been given as a cause. Sometimes the disease follows the exanthemata. Some cases are undoubtedly due to vertebral caries (tuberculosis); in other cases streptococci are found.

Symptomatology: Dysphagia may cause the infant to refuse to nurse. There are cough, slight fever; later dyspnœa and cyanosis. There is a peculiar audible inspiration. In adults there are pain, dysphagia, fever, chills, and dyspnœa. There is bulging upon one side, and palpation will reveal the presence of pus. In adults there is more inflammatory reaction than in children.

Diagnosis: Retropharyngeal abscess should be *differentiated* especially from croup or œdema of the glottis in children by physical examination; and from aneurism and tumors in adults.

Prognosis: Except when due to tuberculosis, the disease usually lasts two to four weeks in children, and in adults runs a course similar to suppurative tonsillitis. Death may be caused by suffocation, erosion of a bloodvessel, rupture into the trachea or œsophagus, septicæmia, or by œdema of the glottis. Sudden death has been attributed to pressure on the large cervical nerves or ganglia.

Retropharyngeal abscess—treatment: Scarification may be practised before there is suppuration. The presence of pus demands its evacuation. Cases due to tubercular caries of the vertebræ are more chronic. It has been recommended that such abscesses should not be opened unless there is danger from some complication or suffocation. When due to tuberculosis, the use of tuberculin and the general treatment of tuberculosis is indicated.

DISEASES OF THE SALIVARY GLANDS.

Mumps has been described under the *Infections*.

The salivary glands may be involved in a suppurative process, to constitute **secondary parotitis**, symptomatic parotitis, or parotid bubo.

The secretion of the salivary glands may be lessened or stopped entirely, constituting the condition known as **xerostomia** or dry mouth.

Or the secretion of the glands may be increased, to constitute **ptyalism** or salivation. The *chief causes* of increased secretion of the salivary glands are pregnancy, certain drugs,

especially pilocarpin, mercury, iodide of potassium, jaborandi, muscarin, and tobacco; certain metals, especially gold, silver, copper, arsenic, and lead; lesions of the pons and medulla; certain infective diseases, especially hydrophobia and smallpox; and some psychic disturbances, especially insanity, hysteria, and the perception of certain tastes.

DISEASES OF THE ŒSOPHAGUS.

GENERAL CONSIDERATIONS.

The diseases: The œsophagus, though freely open to insult, is comparatively *rarely the seat of disease.* Probably one of the most common affections of the œsophagus is *spasm of the œsophagus*, œsophagismus, spasmodic dysphagia, angina convulsiva, or spasmodic stricture of the œsophagus. Paralysis, hyperæsthesia, and anæsthesia of the œsophagus are sometimes encountered. The œsophagus may be affected by organic stricture, carcinoma, dilatation, the formation of a diverticulum, the presence of acute or chronic inflammation or foreign bodies. Perforation and rupture of the œsophagus may occur. Other conditions are ulcer of the œsophagus, hemorrhage, tuberculosis, syphilis, parasitic disease, and congenital defects and malformations.

Inspection may reveal a swelling upon the left side of the neck, due to a diverticulum or tumor of the œsophagus.

Such a revelation may be confirmed by **palpation**, especially when made while the sound is in the œsophagus.

Auscultation may help in the diagnosis of obstruction, especially of the lower end of the œsophagus, since in such cases auscultation practised at the tenth rib upon the left of the spine, or upon the left of the ensiform cartilage, may not disclose the sound produced by the entrance of the bolus of food into the stomach from the œsophagus, which normally occurs about six seconds after swallowing (Meltzer).

Of more practical value is the use of the **sound**, with which we may measure the calibre of the œsophagus, detect and locate strictures and dilatations and sensitive or ulcerated spots as well as foreign bodies. A stomach-tube with a closed

end may be used as a sound. The chief contraindication to the use of the sound is the presence of an aortic aneurism.

Further examination of the œsophagus may be made by means of the **œsophagoscope**, which is rendered more practicable, under cocaine anæsthesia, since the improvements in the electric light. Through the œsophagoscope scars may be seen and portions of suspicious growths removed for microscopic examination. The character of the membrane may also be studied as to the presence of acute or chronic inflammation, and dilatation may be readily recognized (Rosenheim). Foreign bodies, which otherwise may not be removable without the intervention of surgery, may sometimes be withdrawn through an œsophageal tube (Von Hacker).

The *Röntgen ray* is frequently used for the detection of foreign bodies in the œsophagus.

OBSTRUCTION OF THE ŒSOPHAGUS.

Obstruction may be due to strictures, muscular spasm, foreign bodies, tumors, external compression, and congenital stenosis.

Strictures may be caused by caustics, syphilis, ulcer, and cancer. Stricture of the œsophagus is to be differentiated from **muscular spasm**, best by means of the sound.

Foreign bodies usually have a distinct history.

Obstruction of the lumen of the œsophagus may also be caused by a growth of the **thrush fungus** along the œsophageal wall in individuals greatly debilitated.

Tumors, polypi, and cancers may cause obstruction by growing into the lumen of the œsophagus.

External compression of the œsophagus may be caused by prevertebral abscesses and tumors, enlarged cervical and mediastinal lymphatic glands, tumors of the mediastinum and thyroid gland, aneurism, and possibly by a distended diverticulum.

Congenital stenosis is rare, and may be suspected in infancy when the symptoms of obstruction come on without any other apparent cause.

The **prognosis** is good, at least so far as life is concerned, in obstruction of the œsophagus due to *muscular spasm*. *Cica-*

tricial strictures which permit the introduction of the sound may be kept open by the occasional passage of this instrument. Strictures which do not permit the passage of the sound may cause death through marasmus. *Foreign bodies* may be removed. The outlook in *cancer and aneurism* is bad.

Treatment: Foreign bodies should be removed as soon as possible. Strictures require long-continued gradual dilatation. External and internal œsophagotomy have been successful. In cases of obstruction from cancer, careful dilatation secures temporary relief.

DILATATION OF THE ŒSOPHAGUS.

Dilatation may be *caused* by obstruction of the œsophagus, the dilatation appearing above the obstruction. Other cases may be due to a weakening of the muscular wall. Some cases, especially those occurring in early infancy, seem to indicate that the condition may be congenital.

The chief **symptoms** are those referable to the retention of food in the œsophagus. There is dulness to the right of the spine after the ingestion of food; with later regurgitation and vomiting

Diagnosis may sometimes be made clear by the introduction of the *sound.*

DIVERTICULUM OF THE ŒSOPHAGUS.

A circumscribed **dilatation** of the wall of the œsophagus may occur, especially in males, at or before middle life. Zenker and v. Ziemssen believe the condition to be a hernia of the mucous membrane through the muscular coat, weakened by trauma, ulcer, or scar. Some observers believe the condition to be congenital.

Symptoms: Food, which sooner or later must be liquid, may be regurgitated, sometimes to reach the stomach upon being reswallowed. *Regurgitation* may be aided by pressure on the left side of the neck. There is often emaciation. Cases have been found post mortem without symptoms during life.

Diagnosis can usually be made with the *sound.*

Treatment of the diverticulum may be unnecessary. Other cases call for the use of the sound. Extirpation has been successfully performed in several instances (v. Bergmann, Kocher, Butlin, Mixter).

PERFORATION AND RUPTURE OF THE ŒSOPHAGUS.

The œsophagus may suffer **perforation** from foreign bodies, instruments, ulcers; or secondarily, from aneurism, retropharyngeal abscess, tuberculosis of the vertebræ or peribronchial glands, rarely from tuberculosis pulmonum.

The first **symptom** may be pain during swallowing or coughing, sometimes followed by the vomiting of pus (abscess) or of food. The temperature is high and irregular. There is pain upon swallowing. Perforation of a bronchus or of the trachea causes a sudden paroxysmal cough and the ejection of food through the larynx. Later there is severe bronchitis, possibly gangrene and abscess of the lung.

Among the **complications** that may occur are pleurisy, pericarditis, pneumothorax, pyopneumothorax, and pneumopericardium.

The **prognosis** assumes gravity with the severity of the symptoms.

Treatment is symptomatic. Nourishment may be supplied by enemata. Complications, if possible, should be met by surgery.

Rupture of the œsophagus is of rare occurrence. The rupture occurs longitudinally in the front or side of the esophagus, between the bifurcation of the trachea and the diaphragm. There is the history of violent vomiting or efforts to expel a foreign body from the œsophagus. There may be little pain attending the rupture. Later there occur emphysema, pleurisy, and death as a rule within three days, although life may be prolonged a week.

Hemorrhage from the œsophagus may be profuse, from rupture of an aneurism, varicose veins, or from the presence of a foreign body; or the hemorrhage may be slight, from ulcer or cancer. Differentiation from gastric hemorrhage is sometimes difficult. Usually hemorrhage from the œsophagus occurs with regurgitation rather than vomiting. The *treatment* is

symptomatic. If possible any cause, such as a foreign body, should be removed. Ice may be swallowed. Food may be given by enemata. Tannin, gallic acid, and iron are recommended.

INFLAMMATIONS OF THE ŒSOPHAGUS.

Œsophagitis: Inflammation of the œsophagus may be caused by strong alcoholic beverages, corrosive fluids, hot food or drink, foreign bodies, food retained in the œsophagus by obstruction, infectious diseases, especially typhoid fever, scarlatina, variola, tuberculosis, and syphilis; by the extension of inflammation from contiguous structures, and chronic passive congestion due to cardiac obstruction.

The chief **symptoms** are pain on swallowing and regurgitation. Mild cases may show no suggestive symptoms. In severe cases there may be fever, chills, and extreme prostration.

The **diagnosis** in the presence of dysphagia and regurgitation is easy.

Prognosis: Cases of acute catarrh of the œsophagus recover rapidly; cases of chronic catarrh usually show exacerbations and remissions over a long period of time. The prognosis is doubtful in corrosive and suppurative cases because of the tendency to perforation, the formation of fistulæ, gangrene, and stricture.

Treatment: Nourishment may be given per rectum. Ice and cold drinks may be taken internally. Cases caused by acids may receive alkalies, lime-water, or magnesia, to neutralize the acid. Where the cause has been an alkali, dilute acetic acid may be given. Toxic cases are relieved by washing out the stomach, if there be no danger of tearing the œsophagus. Abscesses demand evacuation. The sound- or stomach-tube may be used in cases of chronic œsophagitis, to prevent the formation of a stricture. Pain may call for morphine or cocaine.

Tuberculosis of the œsophagus is rare. The *treatment*, further than the use of tuberculin, is symptomatic.

Syphilis of the œsophagus demands general syphilitic treatment (see Syphilis). Strictures should be treated mechanically, probably best by the passage of the sound.

TUMORS OF THE ŒSOPHAGUS.

Tumors, except carcinomata, are rare.

Carcinoma of the œsophagus is not infrequently encountered. As a rule the cancer is primary. Secondary deposits may occur from cancer of the stomach, more rarely of the pharynx or of the thyroid. About three-fourths of the cases occur in males, usually after forty. It has been observed that drunkards are most frequently affected.

Diagnosis: Evidence of obstruction of the œsophagus, with gradually progressing symptoms, especially in a man past forty, should lead to the suspicion of carcinoma of the œsophagus. About nine-tenths of the cases of obstruction of the œsophagus after forty are due to carcinoma. *Syphilis* may be eliminated by the therapeutic test, if necessary. Pieces may be removed through the œsophagoscope and examined microscopically to make the diagnosis absolute. Sometimes a piece of the tumor, sufficient for microscopic examination, may be discharged by vomiting, or may be removed with the stomach-tube or sound.

Prognosis: The usual duration of life is ten to fifteen months.

Treatment: *Gastrostomy* offers relief in some cases.

ŒSOPHAGISM.

Spasm of the œsophagus occurs most frequently in women, especially in the neurasthenic, hysterical, and hypochondriacal. There are all degrees of dysphagia, and the condition may be of short or long duration, sometimes with intermissions. The *diagnosis* is readily made with the sound. The *prognosis* depends upon the underlying condition.

Treatment: Any local or exciting cause should be removed. The use of the sound is usually the best method of treatment. Attention should be directed to the treatment of the underlying disease—neurasthenia, hysteria, or hypochondriasis.

Angina Ludovici is an acute septic inflammation of the throat, occurring chiefly secondary to the infections, especially diphtheria and scarlet fever, and sometimes caused by trauma. The infectious agent is usually a streptococcus. The submaxillary gland on one side is often first affected.

Treatment should be surgical and thorough.

DISEASES OF THE STOMACH.

ACUTE GASTRIC CATARRH.

Etiology: Acute gastric catarrh is usually due to errors or indiscretions in diet, the use of hot or cold drinks, highly seasoned or fermented foods, insufficient mastication of the food, the ingestion of alcohol and decomposed food, septicæmia, and toxæmia. The disease may be caused by a number of toxic substances: alcohol, phosphorus, arsenic, potassium cyanide, corrosive sublimate, potassium chlorate, concentrated acids, and caustic alkalies.

Symptomatology: Heaviness at the pit of the stomach, later fulness and eructations, constitute the symptoms of the mild form of acute gastric catarrh. Severer cases may show in addition nausea, *pain* in the region of the stomach, headache, fever, vomiting, anorexia, and *constipation* or diarrhœa. The stomach may be *tender and bloated*. The tongue is coated. There may be affection of the gall-bladder and icterus. In cases of *phlegmonous gastritis*, the pain in the region of the stomach is severe, with or without preceding dyspeptic symptoms. The symptoms increase in severity. The fever is high, 103°–105° F. Retching and vomiting of mucus and bile are common. The gastric region is tender to pressure. There is diarrhœa, sometimes constipation.

Toxic cases show symptoms more or less severe according to the cause. There is gastric pain, which becomes more severe upon pressure. There is *vomiting*, sometimes hæmatemesis. There is always thirst. There may develop peritonitis, icterus, and hæmaturia. In severe cases the pulse is weak and there may be cyanosis, cold perspiration, slight coma, possibly collapse and death.

Diagnosis: The *history* is often of value, since there may be revealed the ingestion of improper food or poisons. An *examination of the contents of the stomach* may show a decrease in the amount of the gastric secretions, or the presence of poison in toxic cases.

The diagnosis of *phlegmonous cases* is sometimes difficult or impossible during life. Such cases should be suspected when, in addition to the symptoms mentioned, there are increased resistance in the gastric region and great tenderness upon pressure.

Toxic cases sometimes show affection of the mouth, tongue, or pharynx, due to the ingestion of corrosive or caustic poisons. Such cases may be cleared up sometimes by the history, or positively by the use of the stomach-tube or the examination of vomited matter.

Cases of gastric catarrh, accompanied by fever, should be carefully differentiated from the infectious diseases, which often show an early catarrh.

The *differentiation* between gastric catarrh and *typhoid fever* may be made by the usual sudden onset of the fever in gastric catarrh and the gradual onset of typhoid fever. Herpes is found especially in gastric catarrh; a positive diazo reaction is found in the urine of typhoid fever, and the blood presents the Widal reaction.

The *pain* from *biliary calculi*, as a rule, should not be mistaken for gastric catarrh.

Prognosis: As a rule, the prognosis of simple acute gastric catarrh is favorable. The aged and invalids may suffer serious complications. Phlegmonous cases have a worse outlook, death usually occurring within a week. Some cases may survive two weeks. Cases of so-called gastric abscess may run a chronic course. In the toxic cases the prognosis is doubtful, depending upon the nature and quantity of the poison taken and the condition of the patient when the case comes under treatment.

Acute gastric catarrh—treatment: The stomach should be emptied. This may be accomplished by the use of emetics, best apomorphine hydrochlorate, or in children tartar emetic and ipecac. A better means is the use of the stomach-tube,

with which the stomach may be emptied and thoroughly washed. For a day or two the patient should receive only a light diet—strained gruels and weak tea. The diet may then be gradually increased by the addition of rice, soups, soft-boiled egg; later bread and butter and oysters, until the normal diet is reached. Meat once a day is allowed on the fourth day, if improvement is continuous. Constipation may be relieved by calomel, especially when there is fever, or by seidlitz powders or citrate of magnesium.

In *phlegmonous cases* the treatment is symptomatic. Ice-bags are applied to the abdomen, and opium may be given in large doses, or morphine subcutaneously. Collapse calls for the use of the analeptics, camphor, ether, etc.

In *toxic cases* the treatment varies with the nature of the poison. Strong acids or alkalies should be diluted and neutralized, the former with calcined magnesia and milk, 100 gm. to the pint; the latter with lemonade or a 1 per cent. or 2 per cent. solution of acetic acid. As a rule, the use of the stomach-tube renders the use of antidotes superfluous. In some cases it may not be safe to use the stomach-tube for fear of perforation of the stomach; nor should vomiting be induced, on account of the damage these substances may cause upon again coming in contact with the œsophagus and mouth when ejected. In toxic cases caused by the *alkaloids* and *metals* the use of the stomach-tube is the best treatment. In emergencies emetics may be employed.

Complications call for appropriate treatment.

CHRONIC GASTRIC CATARRH.

Etiology: The chief causes are: improper food, ice-water, and highly-seasoned food; rapid eating, whereby the food is not properly masticated; sore mouth and bad teeth, which may cause trouble also through the swallowing of the products of decomposition; the ingestion of too large quantities of food, the excessive use of tobacco, chewing or smoking; the drinking of tea and coffee, and alcoholic beverages, especially the stronger ones, and the use of so-called "stomach bitters."

Chronic gastric catarrh may follow repeated attacks of acute

gastric catarrh, or date its origin from an attack of some severe infection, such as typhoid fever. Some cases are secondary to certain diseases, such as gout, diabetes, and chronic diseases of the lungs, heart, liver, and kidneys.

Chronic gastric catarrh—symptomatology: The onset is insidious. There are a bad taste in the mouth, poor appetite, a feeling of fulness and oppression after meals, sometimes palpitation and shortness of breath, and possibly dizziness, which may compel the patient to seek the recumbent posture. Belching is frequent; and often there are regurgitation, sometimes nausea, less frequently vomiting. Constipation is the rule; or there may be diarrhœa, and in some cases constipation and diarrhœa alternate. The urine is scanty and frequently contains phosphates and urates. There are languor, lack of energy, often headache, in some cases persistent yawning and inability to breathe deeply. There may or may not be loss of weight. The tongue is coated and indented by the teeth. The gastric region is bloated and tender upon pressure.

Diagnosis: The gradual onset and chronic course of the disease, with the symptoms enumerated, should lead one strongly to suspect chronic gastric catarrh. The diagnosis may be confirmed by an examination of the contents of the stomach. The patient may be given the *test-breakfast* of Ewald, consisting of an ordinary roll or piece of white bread without butter, and a large cup of tea without sugar or milk. The stomach-contents are withdrawn one hour later. The quantity of the contents will vary from 120 to 180 c.c., and the roll will not be found reduced to as fine particles as when digestion is normal. Mucus is present in great quantities in some cases, and absent in others. In the presence of mucus, acetic acid produces turbidity when added to the filtrate. Free hydrochloric acid is altogether absent or present in only small quantities. Pepsin and rennet may be found. There are small quantities of erythrodextrin, and an abundance of achroödextrin and sugar. The absorptive power of the stomach does not necessarily present any considerable alteration from normal.

The **differential diagnosis** concerns chiefly cancer, the neuroses, and achylia gastrica. Pain, hæmatemesis, and marked

emaciation are absent in chronic gastric catarrh, and present in ulcer and cancer. The gastric neuroses may be recognized by the presence of other nervous symptoms. The symptoms and stomach-contents show less variation in chronic gastric catarrh than in the gastric neuroses. Achylia gastrica shows not only absence of hydrochloric acid, but also absence of rennet and pepsin.

Prognosis: Good, but recovery is tedious. Recurrences may be caused by indiscretions in diet.

Chronic gastric catarrh—treatment: The *diet* should be regulated, and at first light, consisting chiefly of milk, kumyss, matzoon, soups of barley, oatmeal and rice in milk, chicken soup, which may contain an egg, soft-boiled eggs, mashed potatoes, scraped meat, toast; later bread and butter, tea and cocoa. The patient should avoid meat that is tough, too fresh or too fat, especially pork, sausages, and lobster, salmon, chicken salad, mayonnaise, cucumbers, pickles, cabbage, and alcoholic beverages. The meals must be eaten slowly and the food thoroughly masticated.

The patient should observe regular hours and exercise especially in the open air, walking, driving, riding, rowing. Ten minutes' gymnastics may be taken every morning, followed by a cold sponge-bath and thorough rubbing. Ventilation with pure air is important.

Further than the regulation of the *diet* and *habits*, most may be accomplished by *lavage*. The stomach should be washed out before breakfast every other day for two or three weeks.

Electricity may be used, a large sponge-electrode placed over the gastric region and a smaller electrode upon the back, about the level of the seventh vertebra, on the left of the spine. A better method is the employment of intragastric electrization by means of a deglutable stomach electrode (Einhorn), using the faradic current. Such treatment should be continued two or three months.

Certain *mineral waters* are excellent therapeutic agents in chronic gastric catarrh, and are best taken at the springs, since there the patient is placed under the best hygienic surroundings. A tumblerful is taken upon arising. As a rule,

the saline waters, containing sodium chloride with varying amounts of carbonic acid gas, are to be preferred. In cases of marked constipation, waters containing sodium sulphate, sodium carbonate, and sodium chloride, with large amounts of carbonic acid gas, are useful.

Of *drugs*, hydrochloric acid is especially indicated by its deficiency in the gastric juice. Ewald recommends gtt. xl-lx of dilute hydrochloric acid three times a day. Einhorn gives smaller doses, gtt. vj–xij, of dilute hydrochloric acid in a glassful of water half an hour after meals, taken "one-third at a time at intervals of a quarter or half an hour." Pepsin is rarely useful. Of more value are the bitter tonics, condurango, quassia, gentian, kino, columba, and nux vomica, taken fifteen minutes before meals in a wineglass of water. Creosote is of value in the gastritis of phthisis.

Constipation may be relieved largely by regular habits of going to the closet. The ingestion of coarse foods increases the quantity of fæces and peristalsis. Thus rye bread and green vegetables, such as spinach, asparagus, and green peas, are useful. The diet may also include fruits, cooked pears, stewed or baked apples, and stewed prunes. A good combination is two parts of prunes and one part of dried figs (Ewald). An orange may be eaten in the morning. A glass of water or milk may be taken upon arising. In some cases rhubarb or cascara sagrada will be found useful. Severe constipation calls for aloes and podophyllin. Einhorn recommends:

℞. Podophyllin, gr. v (0.3);
Ext. nucis vomicæ,
Ext. calabar bean, āā gr. viiss (0.5);
Ext. gentian.,
Ext. pulv. glycyrrh., āā q. s.
M. et ft. pil. No. 30.
S. One pill twice a day.

Enemata are useful, especially in cases of atony or weakness of the large intestine. For an enema there may be used one quart of lukewarm water, containing a teaspoonful of

common salt. The injections should be given daily, always at the same time, for two weeks. Glycerin suppositories or injections of glycerin in water, a teaspoonful to four or five tablespoonfuls of water, are sometimes useful.

GASTRIC ULCER.

Etiology: *Ulcer of the stomach* is found in 5 per cent. of post-mortems, about twice as frequently in women as in men (Brinton); most often between twenty and forty years of age (Ewald).

The cause is unknown; but many theories have been formulated, some of which have been more or less generally accepted.

V. Sohlern, who found the inhabitants of certain parts of Germany and Russia practically exempt from the disease, attributed this fact to an exclusive *vegetable diet* and recommended such a diet and the administration of some of the salts in prophylaxis.

It has been frequently asserted, but not without contradiction (Ewald), that ulcer of the stomach is especially frequent in certain *vocations* (shoemakers).

Experimental injuries of the gastric mucous membrane in animals heal readily (Griffini and Vassale), except when there is some *alteration in the blood*, anæmia (Quincke and Daettwyler); hæmoglobinæmia (Silbermann). Gastric ulcer is especially frequent in *chlorotic patients*.

The introduction of dilute hydrochloric acid into the stomach (0.5 per cent. solution), after the production of gastric hemorrhage by section of the spinal cord, caused the formation of deep ulcers (Koch and Ewald).

The formation of gastric ulcer has been attributed to the *destruction of an infarct* caused by an embolus or thrombus (Virchow), and ulcer has been produced in this way experimentally by Panum. We are not always able to find an embolus or thrombus in cases of gastric ulcer.

Pavy attributed the formation of ulcer of the stomach to a *diminished alkalinity* of the blood permitting the tissue to be attacked by the gastric juice. It is now believed that *hyper-*

acidity of the gastric juice plays an important rôle in the etiology of gastric ulcer. Chlorosis, anæmia, and amenorrhœa are frequently associated with ulcer of the stomach, and in these conditions there is, as a rule, hyperacidity of the gastric juice. At any rate, it is well known that hyperacidity of the gastric juice is usually found in cases of ulcer of the stomach; but Einhorn has observed ulcer of the stomach in cases showing an "entire absence of gastric juice."

Gastric ulcer—symptomatology: There is early disturbance of digestion; at first epigastric uneasiness and pain; later, nausea, regurgitation, and vomiting. There may be hemorrhage. The disturbances of digestion and hemorrhage cause anæmia and cachexia.

As a rule, *pain* comes on in from two to ten minutes *after the ingestion of food*, and continues as long as there is food in the stomach. Exceptionally pain appears only half an hour to two or three hours after the ingestion of food. Pain is increased especially by coarse and indigestible food, and is more likely to be severe after a large meal. The *pain or tenderness* in the epigastrium is *circumscribed*. After a few weeks or months pain appears in the dorsal region, usually to the left of the eighth or ninth dorsal vertebra. *Hemorrhage* may be revealed by hæmatemesis or melæna; or in very severe cases the blood may not escape before death and be found in the stomach upon autopsy. Small hemorrhages may be detected only upon microscopic or chemic examination of the stomach-contents. The *appetite is good;* but patients may refuse to eat for fear of pain.

Diagnosis: Cases of ulcer of the stomach often show hyperchlorhydria. Useful points in diagnosis are the *age* and *sex* of the patient; the localization, character, and time of occurrence of *pain;* the *appetite*, regurgitation, and vomiting; the increased quantity of *gastric juice*, absence of lactic acid, and, as a rule, increase of hydrochloric acid; hæmatemesis, perforation, and anæmia. Some idea as to the location of the ulcer may be obtained from the position assumed by the patient for the relief of pain, since most comfort will probably be experienced when the ulcer is brought least in contact with the gastric juice.

Ulcer of the stomach should be *differentiated* especially from gastralgia, hyperchlorhydria, and gastric carcinoma.

Prognosis should be guarded. The outlook is better in cases that come under treatment early.

Gastric ulcer—treatment: Rest in bed should be observed for two or three weeks. Milk is the best food, to which may be added barley-water, oatmeal-water, rice-water, plain water, weak tea, peptone, meat-powder, and lactose. During the first week the patient should receive half a glassful of milk every hour. During the second week the quantity is increased to a glassful and a half every two hours, and the patient may receive once or twice a day a raw egg beaten up in the milk. The third week the patient may receive, in addition, barley, farina, rice, soft-boiled eggs, and crackers and milk, every three hours. During the latter half of the third week meat may be added, at first raw scraped meat, later broiled. Gradually the regular diet is resumed. During the third week the patient may get up, at first for a short time only, and may go out of doors the fourth week and gradually resume the ordinary daily routine of life. During the first two weeks warm flaxseed poultices may be applied over the region of the stomach during the day, and a wet linen cloth at night.

Severe pain, vomiting, or hæmatemesis may necessitate rectal alimentation. In the morning the intestine should be washed out, best with a quart of water containing a teaspoonful of common salt. An hour later an enæma may be given consisting of one or two raw eggs beaten up in a glass of milk, to which a pinch of salt is added; or a teaspoonful of peptone in a cup of water. The enema should be given at a temperature of about 100° F., and repeated three or four times a day. This may be continued five days, when food may usually be given per os.

Pain may be relieved by codeine. Constipation is controlled by Carlsbad salt. Hyperacidity of the gastric juice may be relieved by:

℞. Magnesiæ ustæ, ʒj (5.0);
Sodii carbonatis,
Sodii bicarbonatis,
Elæosacch. menth. pip., *āā* ʒiij (15.0).
M. exactissime, f. pulv.
S. As much as will rest on the point of a knife, every two hours. (Einhorn).

Patients who cannot remain in bed may receive nitrate of silver, 0.3–0.6 : 180.0 water, a tablespoonful in a wineglassful of water half an hour before meals. Or subnitrate of bismuth may be used, 3.0-5.0 in a wineglassful of water, half an hour before meals. Such treatment should be continued two or three weeks.

Hemorrhage is met by the application of an ice-bag over the region of the stomach and absolute rest in bed. In severe cases ergot may be given subcutaneously, or the stomach may be carefully washed out with ice-cold water (Ewald), best after the application of cocaine to the pharynx to prevent excessive retching.

For *collapse*, the analeptics, best camphor and ether, may be used subcutaneously. A hot-water bag should be placed at the feet, and there may be given an enema of warm wine, to which an egg may be added. Often salt-water infusion is of the greatest value.

Perforation demands absolute rest and abstinence from food and drink, the use of the ice-bag over the stomach, and of opium, best in suppositories. Should there be a large quantity of food in the stomach, it may be carefully removed with a stomach-tube. Laparotomy and suture have been successful in some cases.

GASTRIC CARCINOMA (Carcinoma Ventriculi; Cancer of the Stomach).

Etiology: No specific infectious agent has been isolated. Carcinoma is believed by Pfeiffer to be due to a variety of the sporozoa. The stomach, with the possible exception of the uterus, is the most frequent seat of cancer, about one-fourth

of all cases of cancer occurring in the stomach. The occurrence of the disease seems on the increase. The age of greatest liability is forty to sixty years. But congenital cases have been reported (Wilkinson and Wiederhoefer). Sex seems to exert little or no influence. Heredity in some cases seems to play a rôle in etiology. Trauma, mental worry and anxiety, and indulgence in cider and sour wines have been considered causes.

The **chief varieties**, in the order of frequency, are: scirrhous, medullary, colloid, melanotic, and epithelial carcinoma.

Cancer of the stomach—symptomatology: Usually there is *anorexia;* in 85 per cent. of cases (Brinton). There may be an aversion for certain foods, especially meat and albuminous food, and a craving for highly-seasoned food, pickles, herring, etc. *Pain* is present in almost all cases, and is more or less constant, frequently of a lancinating character, sometimes dull, gnawing, burning. There may be tenderness upon pressure. The ingestion of food causes little if any increase of pain, and there is no relief afforded by vomiting. *Vomiting* occurs in perhaps even more cases than anorexia, oftener when the carcinoma is at the pyloric or cardiac extremity of the stomach than when situated elsewhere. Hæmatemesis is present in many cases, sometimes melæna.

A *tumor*, usually hard, irregular, nodulated, sometimes smooth and small, may be revealed by gastrodiaphanoscopy sometimes before the diagnosis can be made by palpation. Most cases show cachexia.

Examination of the blood reveals a reduction of red blood-corpuscles and hæmoglobin, and an increase of leucocytes. Probably of more importance is the observation that the number of leucocytes is about the same at the height of digestion as during fasting (Schneyer). Normally the number is increased during digestion.

The *urine* may contain more nitrogen than is introduced with the food (Klemperer, Müller). Frequently there is a diminution of the chlorides; occasionally there is peptonuria, indicative of absorption from some ulceration in the digestive tract.

Carcinoma at the *cardiac extremity* of the stomach usually

causes dysphagia and may produce complete stenosis. The deglutition-sound, made by the bolus of food entering the stomach, may be absent or retarded, appearing sometimes twenty seconds, instead of seven seconds, after swallowing. Examination with the stomach-tube or sound may reveal more or less diminution of the calibre of the cardiac opening. Sometimes particles of the tumor may be withdrawn through the stomach-tube. These should be examined microscopically. The constant presence of blood upon examination with the stomach-tube should raise the suspicion of cancer, probably situated at the cardiac extremity of the stomach.

Carcinoma at the *pyloric extremity* of the stomach causes pain, a feeling of fulness, and vomiting of chyme and of food which may have been taken a day or two before. The tumor is usually to the right of the linea alba, between the umbilicus and ribs.

Carcinoma of the *stomach proper* presents a constant gnawing pain, marked cachexia, tumor to the left of the linea alba, vomiting of food, and the retention of chyme in small quantities.

Diagnosis: The great majority of cases of cancer of the stomach occur after forty. Pain, usually not intense, is continuous. The appetite is poor; the tongue usually heavily coated. Eructations are the rule, the odor often being disagreeable, sometimes fœtid. There is pyrosis. Vomiting, often of large quantities, occurs once or twice a day or every other day. The gastric juice is usually greatly decreased in amount. The blood discharged by hæmatemesis is usually slight, as a rule coffee-brown in color, often decomposed and fœtid.

Palpation will usually detect a tumor, movable, uneven, tender to pressure. Perforation occurs only late in the disease. Cachexia is marked.

The *examination of the gastric juice* reveals an absence of free hydrochloric acid and the presence of lactic acid. Rarely, free hydrochloric acid may be found in carcinoma, and lactic acid has been found in non-malignant cases.

A *microscopic examination* of the particles withdrawn from the stomach may make the diagnosis absolutely positive.

The *differential diagnosis* has to do with *ulcer*, benign stenosis of the pylorus, *chronic gastric catarrh*, achylia gastrica, severe gastric neurasthenia, and benign tumors.

Prognosis: Death may occur within a month to two years, usually within a year, depending upon the situation and variety of the carcinoma and the complications that may arise.

Cancer of stomach—treatment: Good temporary results have been reported from the use of the iodides, arsenic, condurango, and a great number of remedies. Pain may be relieved by opium, morphine, codeine (usually best given in combination with belladonna or atropine). Constipation is relieved by rhubarb, compound licorice powder, cascara sagrada, enemata, glycerin suppositories. Obstinate vomiting may be obviated by washing out the stomach occasionally or by the use of opiates. Hemorrhage, when considerable, may be treated as in cases of ulcer of the stomach. When there is decomposition of food or ulceration, chloral may be advantageously given (Ewald, Einhorn). The patient should receive an abundance of such food as he can take and assimilate.

In the way of absolute cure, more hope is offered by surgery. Complete removal of the carcinoma, by resection of the pylorus, excision of the tumor, or even removal of the stomach, may be resorted to when the diagnosis is made early, before the tumor has attained a large size, and before metastases have formed. Other contra-indications are great anæmia or cachexia and extreme age. Gastrostomy in carcinoma of the cardia or œsophagus, and gastro-enterostomy in carcinoma of the pylorus, may be resorted to as palliative measures.

GASTRECTASIA.

Gastrectasia, dilatation of the stomach, is *caused* most frequently by obstruction of the pylorus. Thus the condition occurs most frequently in carcinoma and from the cicatrization of ulcers. Less frequently gastrectasia may be caused by paresis of the stomach-wall, the result of chronic catarrh

of the stomach. Sometimes the condition is due to fatty degeneration, extensive atheroma, or amyloid disease. Rarely there may be no apparent cause.

Symptomatology: The stomach is increased in size, so that the fundus occupies the left hypochondrium, the pylorus extends into the right hypochondrium, and the lower border of the stomach may be found below the umbilicus. The condition is frequently revealed by the large quantity of material discharged during the act of vomiting. Digestion is delayed, and the stomach-contents show evidence of decomposition, acetic and butyric fermentation, and the presence of sarcinæ and various other micro-organisms. Nervous symptoms are common, especially depression of spirits and hypochondriasis. Sometimes there is coma.

Prognosis: The outlook depends upon the cause. Cases due to paresis depending upon catarrh of the stomach may recover. The prognosis is bad in carcinoma.

Treatment: Most may be accomplished by lavage and diet, both in the cure and relief of the condition. Digestion may be aided by the use of dilute hydrochloric acid and the bitter tonics, especially the tincture of nux vomica. Faradization is often of value. Constipation may be relieved best by Carlsbad salts, the compound pill of rhubarb, or cascara sagrada. Carcinoma may justify an appeal to surgery. There are also cases of simple gastrectasia which have been benefited by placing a *reef* in the wall of the stomach (gastrorrhaphy).

GASTROPTOSIS.

Gastroptosis is a downward displacement of the stomach, frequently associated with displacement of other organs, especially with enteroptosis, nephroptosis, and hepatoptosis. The prolapse of the abdominal viscera may become general, to constitute a splanchnoptosis.

Symptoms: There are weakness, lassitude, sleeplessness, and constipation or irregularity of the bowels. The patient experiences difficulty in the digestion of fats, farinaceous foods, acids, pure wine, pure milk, with an increase in the digestive troubles about three hours after meals (Einhorn). Physical

examination reveals decreased abdominal tension and prolapse of the abdominal organs.

Diagnosis is best made by inflation of the stomach or the use of the gastrodiaphane.

Treatment: The intestines and stomach should be raised and the abdomen supported by a well-fitting abdominal bandage. Constipation must be corrected. Further treatment is symptomatic, and depends largely upon the result of the examination of the contents of the stomach. The bowels should be regulated. Deficient secretion may be stimulated by massage, electricity, and lavage.

FUNCTIONAL DISTURBANCES OF THE STOMACH.

In **hyperchlorhydria** the amount of acid and ferments, and sometimes the total amount of the gastric juice, are increased. There is usually pain two or three hours after meals, relieved especially by albuminous food and alkalies. The appetite may be increased above normal. As a rule, there is constipation.

Treatment, which is symptomatic, calls for the avoidance of mental work and regulation of the daily life. Acids and spices should be excluded from the diet, which should consist largely of albuminous food. After meals the patient must rest fifteen or twenty minutes. Alkalies are indicated. In the absence of constipation, bicarbonate of sodium and magnesia usta may be given. When there is constipation, Einhorn recommends:

℞. Magnesiæ ustæ,
Pulv. rad. rhei, āā ʒij (7.5).
Sodii carbon. exsiccat.,
Sodii bicarbonatis,
Elæosacch. menth. pip., āā ʒiv (15.0).
M. exactissime, f. pulv. D. ad scatulum.
S. Half a teaspoonful to a teaspoonful three times daily, two hours after meals, to be taken in plain water or Vichy water.

Gastrosuccorrhœa continua periodica is characterized by a periodic continuous flow of gastric juice, with vomiting and severe pain.

Gastrosuccorhœa continua chronica shows a constant secretion of gastric juice even when the patient is fasting; and runs a chronic course. Cases of gastrosuccorrhœa always show *hyperchlorhydria.*

In the **treatment,** liquids must not be allowed in large quantities. Lavage is of value, either with plain water or with solutions of nitrate of silver, 300 c.c. of a 1 : 1000 or 2 : 1000 solution (Reichmann); or the stomach may be sprayed with a similar solution of nitrate of silver (Einhorn). Einhorn also recommends direct galvanization of the stomach. Atropine, gr. $\frac{1}{32}$ daily (Voinovitch), or sulphate of morphine, gr. $\frac{1}{3}$–$\frac{1}{2}$ three times a day (Leubuscher, Schaeffer), may be used temporarily.

In **achylia gastrica** the gastric juice is not formed. A positive diagnosis is based upon repeated examination of the stomach-contents and the exclusion of cancer. The small intestine perfectly replaces the digestive work of the stomach, and the organism is not only enabled to maintain its equilibrium, but also to gain in weight (Einhorn).

In the way of **treatment,** the stomach may be stimulated by lavage, direct faradization, and the use of condurango or nux vomica. The food should be thoroughly masticated, that it may be more easily digested in the intestine.

Ischochymia is marked by a retention of food (chyme) in the stomach, even in the fasting condition. The condition may be due to a lessening of the motor function of the stomach, or to stenosis of the pylorus, organic or spasmodic.

In **diagnosis,** most important is the differentiation between benign and malignant stenosis of the pylorus, which may be made by the duration and course of the disease, the presence or absence of tumor, and the examination of the contents of the stomach as to odor, acidity, and the presence or absence of free hydrochloric acid, lactic acid, and rennet.

The **treatment** depends largely upon the cause. Lavage and

spraying the stomach with nitrate of silver, 1 : 1000 to 3 : 1000, are recommended. Malnutrition may call for rectal alimentation.

GASTRIC NEUROSES: SENSORY.

Hyperorexia: Bulimia (ox-hunger), cynorexia (dog-hunger), is marked by a great increase of hunger.

The *diagnosis* of hyperorexia as a primary affection should be based upon the abnormal hunger and the exclusion of other affections to which such a condition may be secondary, such as ulcer of the stomach, hyperchlorhydria, carcinoma of the stomach, intestinal troubles, tapeworm, diabetes, Graves' disease, hysteria, neurasthenia, and tumors of the brain.

In *treatment*, food should be administered in small quantities every two hours. The bromides may be given in large doses twice a day; or cocaine (Rosenthal) 3-5 cgm. twice a day; or opium or codeine 3-4 cgm. three times a day; or arsenic, Fowler's solution, two or three drops two or three times a day. Severe and persistent cases call for a change of climate, to the mountains or seashore.

Parorexia, perversion of appetite, may occur as *malacia*, a craving for highly seasoned food and spices, mustard, salad, vinegar, green fruits, etc.; *pica*, a desire to eat substances not commonly used as food, such as coal, ashes, chalk, earth, sand, insects, etc.; and *allotriophagia*, in which the appetite calls for disgusting and harmful substances, fæcal matter, needles, pins, etc. Malacia may occur in many disturbances of the stomach and in neurasthenia; pica and allotriophagia are found in bad cases of hysteria, more frequently in idiots and the insane.

Polyphagia is a condition in which abnormal quantities of food are required to secure a feeling of satiety. The condition may occur as a primary affection in neurotic individuals. As a secondary affection, polyphagia occurs especially in carcinoma of the pancreas and spleen, fistulous openings of the gall-bladder, diabetes, in some cases of brain-tumors, and in the conditions in which we find hyperorexia as a secondary affection.

Akoria, a condition in which a feeling of satiety may not be experienced, is found especially in neurasthenia and hysteria, and in the conditions in which hyperorexia and polyphagia occur secondarily. The condition often occurs in association with polyphagia.

Anorexia, an absence of hunger and appetite, occurs in many organic and so-called functional disorders of the stomach. A diagnosis of nervous anorexia involves the exclusion of organic affections, cancer, tuberculosis, etc., in which anorexia may occur as a secondary condition.

A valuable point in the *diagnosis* of nervous anorexia is the fact that the loss of appetite causes no alarm on the patient's part.

In the *treatment* of mild cases it is sufficient to offer the patient nourishment regularly and in ample variety, without previous reference to the diet, and then to impress upon him the importance of eating. The patient may receive, fifteen or twenty minutes before meals, tincture of nux vomica, gtt. x three times a day, or fluid extract of condurango, gtt. xx three times a day, or fluid extract of Peruvian bark a teaspoonful three times a day, or orexicum basicum gr. iij-ivss three times a day. More severe cases call for isolation and the "rest cure," with massage and electricity. Persistent refusal of food may demand forced feeding, gavage, through the stomach-tube. Iron and arsenic, Fowler's solution or Roncegno water, may be advantageously administered. Exercise in the fresh air is a valuable tonic.

Gastric hyperæsthesia, apart from gastric catarrh, erosions, ulcer, and carcinoma, occurs especially after chlorosis, excess in venery, and persistent indulgence in improper food.

Treatment as far as possible should address the underlying condition. Chlorotic cases are benefited by iron. In other cases, as a rule, most may be accomplished by the use of the bromides.

Gastralgia, pain in the stomach, more or less severe, periodic in character, may occur in connection with ulcer, cancer, hyperchlorhydria, adhesions of the peritoneum, or after the

ingestion of certain foods or spices to which the patient is not accustomed.

Some cases are distinctly of *central* origin, found especially in connection with spinal disorders, tabes dorsalis, subacute myelitis, pressure-myelitis; much less frequently diseases of the brain, and sclerotic degeneration of the nucleus or trunk of the vagus.

Gastralgia often occurs in hysteria and neurasthenia, and in the infections, especially malaria.

Among the *intoxications* that may cause gastralgia are chronic lead-poisoning, the excessive use of mercury, and the use of tobacco. Cases may be caused by gout, anæmia, and chlorosis.

Among the *reflex* causes are diseases of the genito-urinary organs, displacements of various abdominal organs, and hydronephrosis.

The *diagnosis* of *nervous gastralgia* demands the exclusion of chronic gastric catarrh, carcinoma of the stomach, ulcer of the stomach, stenosis of the pylorus, hyperchlorhydria, gastrosuccorrhœa continua periodica and chronica, achylia gastrica, and conditions in which the pain is outside of the stomach, such as muscular pain due to rheumatism or over-exertion, intercostal neuralgia, gall-stones, kidney-stones, and enteralgia.

Treatment, when possible, should address the underlying condition. The attacks should be relieved by hot applications and drinks. Intense pain may demand morphine hypodermatically, or codeine or opium with belladonna or atropine in suppositories.

GASTRIC NEUROSES: MOTOR.

Cardiospasmus, a spasm of the cardia, may be either acute or chronic. The condition should be differentiated from organic stricture, best by the use of the œsophageal sound or stomach-tube. Often a large sound or tube will enter more readily than a small one.

Treatment. Acute cases call for the bromides and the regular use of the sound. In chronic cases the food must be at first fluid or semifluid, and the stomach-tube should be used

every evening before retiring. Later the usual diet may be gradually resumed.

Eructation is a belching of gas. *Pyrosis*, "heartburn," is an ejection of some of the contents of the stomach into the œsophagus. *Regurgitation* implies an ejection of the food from the stomach into the mouth. *Rumination* is commonly known as "chewing the cud."

Nervous vomiting can be said to exist only after a careful attempt to exclude abnormal conditions of the stomach or of the food. Among the *causes* of nervous vomiting are spinal or cerebral irritation, neurasthenia, hysteria, and diseases of distant organs which may act reflexly.

Pneumatosis, distention of the stomach with gas, is frequently associated with neurasthenia and hysteria. Organic affections must be excluded.

Hyponakinesis ventriculi: Reduction of the mechanical power of the stomach.

Hyperanakinesis ventriculi: Exaggeration of the mechanical function of the stomach. When the peristalsis of the stomach becomes visible upon inspection, the condition is known as *peristaltic restlessness* when the waves proceed from left to right; and *antiperistaltic restlessness* when the waves move from right to left.

Pyloric incontinence may be caused by neoplasms or atony of the pylorus. The condition is recognized especially by regurgitation of the intestinal contents, bile, into the stomach in considerable quantities, which may be revealed by the stomach-tube. The rapid passage of food from the stomach into the intestine, so that the stomach is found empty sooner than normal, may be due to incontinence of the pylorus, but more frequently is caused by *hyperprochoresis*, increased motor function.

Pylorospasmus, spasm of the pylorus, may occur independently of demonstrable organic disease.

Atony of the stomach refers especially to a weakened or retarded muscular action. The condition is found most frequently in chronic gastric catarrh, hyperchlorhydria, neurasthenia, tuberculosis, and heart-disease.

Sometimes atony occurs as a *primary neurosis*, the symptoms of which are fulness after meals, eructations of gas, diminished appetite, and frequently headache and constipation.

The *treatment* calls for intragastric faradization (Einhorn), strychnine, tincture of nux vomica, fluid extract of condurango, and iron. The diet should be light and contain little fluid. Mental work and anxiety must be avoided. Outdoor exercise is of value. Constipation should be remedied.

GASTRIC NEUROSES: SECRETORY.

It is well known that the **gastric secretion** may be increased by the sight of food, and decreased by fear and anxiety. Disturbances of secretion, *hyperchlorhydria*, *hypochlorhydria*, or *achylia gastrica*, may be of *nervous origin*.

Nervous dyspepsia (Leube), neurasthenia gastrica (Ewald), is a condition in which there are dyspeptic symptoms in the absence of any demonstrable organic lesion, when on examination the secretion of gastric juice is found to be normal and the stomach is empty seven hours after a test-dinner.

Etiology: Nervous dyspepsia frequently forms a part of neurasthenia and hysteria, and is found often in chlorosis, diseases of the lungs (tuberculosis), the infections (malaria), diseases of the genito-urinary organs, and in cases of sexual excess and abuse of tobacco and alcohol.

Symptomatology: The appetite is capricious. The tongue is usually clean, sometimes coated. During gastric digestion there are slight pain in the stomach, belching, sometimes drowsiness and headache. When the stomach is empty there may be a feeling of weakness and dizziness. Usually there is depression of spirits. Later the symptoms become aggravated, and there is loss of weight.

The **diagnosis** depends upon the presence of dyspeptic symptoms and the absence of organic disease. A useful point in

diagnosis is the fact that the symptoms are not affected by the character of food ingested, but may be relieved by change of climate and pleasant mental emotions.

The *differential diagnosis* concerns especially chronic gastric catarrh, ulcer, and carcinoma.

Treatment: Existing ailments which may have an etiological relation to *nervous dyspepsia* should receive proper attention. Change of climate, especially a change from indoor to outdoor life, relief from mental anxiety and business and family cares, the ingestion of plenty of wholesome food, and the "rest cure," give the best results. The nervous system may be strengthened by mild hydrotherapeutics, massage, electricity, and moderate gymnastics. Of medicines, the bromides are largely used. Iron, arsenic, nux vomica, and basic orexin are often of value. Sleeplessness may be overcome with chloral, sulfonal, or trional. The bowels should be regulated.

Condition of the stomach in diseases of other organs: Disturbance on the part of the stomach is especially marked in pulmonary tuberculosis, sepsis, chlorosis, and anæmia; diseases of the heart, liver, or kidneys; diabetes, arthritis deformans, gout, and malaria.

DISEASES OF THE INTESTINES.

INTESTINAL CATARRH (Enteritis (Inflammation of the Small Intestine); Colitis (Inflammation of the Large Intestine); Entero-colitis (Inflammation involving both the Small and Large Intestine)).

Localized inflammations have received special names: duodenitis, typhlitis, proctitis, etc.

Catarrh of the intestine may be either *acute* or *chronic*.

Etiology: *Acute catarrh of the intestine* may be caused by errors in diet, especially the ingestion of spoiled food, tainted meat, fruit, milk, bad water, or by irritant substances, organic (drastic cathartics, colocynth, croton oil, carbolic acid) or inorganic (metal poisons, arsenic, corrosive sublimate, tartar emetic, and the caustic alkalies). Bacteria play a prominent rôle in the etiology of this disease. Many cases are attributed

to "catching cold." Among the mechanical causes are enteroliths, gall-stones, and intestinal parasites. An acute duodenitis may be caused by burns of the skin, probably through the action of toxins. Catarrh of the intestine may be caused secondarily by diseases of the mouth or stomach, acute infectious diseases, chronic cachexias, heart-disease, kidney-disease, tuberculosis pulmonum or intestinalis, diabetes, strangulation, volvulus, invagination, peritonitis, thrombosis, and embolism.

Streptococcus enteritis is often complicated by septic invasion of other organs, especially the liver, kidneys, spleen, and heart.

Chronic catarrh of the intestine may be primary or follow an acute catarrh through continuance or repetition of the cause. Most cases depend upon ulcerative processes: typhoid fever, tuberculosis, dysentery, carcinoma, and syphilis.

Symptomatology: *Acute catarrh of the intestine* usually begins suddenly with *pain* (colic) and *diarrhœa*. The diarrhœa is caused by increased peristalsis and decreased absorption. Mucus and fluid are excreted by the intestine. There are from two or three to twenty or more stools in the twenty-four hours. There may be tenesmus when the catarrh extends to the large intestine. The stools are at first fæcal; then thin, later watery. Sometimes there is no diarrhœa, through absorption of fluid in the large intestine. Bile-pigment is frequently present in the stools, especially when the catarrh involves the upper part of the small intestine. The stools of infants and small children are, or become on standing, green from oxidation of the bile-pigment. Sometimes there is *icterus*. There are *borborygmus and meteorism*. Upward pressure of the diaphragm may cause dyspnœa, palpitation, and precordial distress. There are early *malaise and prostration*. Fever is usually absent, but may reach 102° F. Convulsions or delirium may supervene in weak individuals, especially children and the aged. The loss of considerable fluid through diarrhœa causes *diminution of the urine*, sometimes anuria, and increased *thirst*. There are anorexia and nausea, except when the catarrh is limited to the large intestine. Burning, itching, pain, and tenesmus may be distressing in catarrh of the rectum.

In *chronic catarrh of the intestine*, diarrhœa may alternate with *constipation*. Constipation is more frequent than continuous diarrhœa. Tenesmus often causes hemorrhoids. *Mucus* is always present, and sometimes may be passed in large quantities. Sometimes the stools contain *blood* and *pus*. A proctitis may extend to constitute a periproctitis, with the formation of abscess, which by bursting may cause an external rectal, recto-vesical, or recto-vaginal fistula. The subjective symptoms of chronic catarrh are similar to those of acute catarrh. The general symptoms may be more pronounced. The mucous membrane is sometimes destroyed, or undergoes atrophy, to cause persistent chronic diarrhœa, anæmia, and debility. When this is combined with atrophy of the stomach, there may be the picture of pernicious anæmia (Osler, Nothnagel, Ewald).

Diagnosis: *Catarrh of the duodenum* is often caused by extensive burns of the skin. The condition may be recognized by the presence of icterus, tenderness in the right hypochondrium following catarrh of the stomach, and the passage of large quantities of mucus.

Catarrh of the small intestine shows indican in the urine, which may be recognized by the Burgundy-red reaction of Rosenbach. Boil the urine in a test-tube, adding nitric acid drop by drop. A Burgundy or peony-red color, which is retained on further boiling and which may be extracted with ether, is indicative of a disturbance of the metabolic processes in the small intestine.

Catarrh of the large intestine, especially of the rectum, is indicated by anal itching and burning sensations, tenesmus, pain in the left iliac fossa, the passage of mucus, sometimes of blood, and considerable pain on digital examination, especially when the lower part of the large intestine is affected.

Chronic catarrh of the intestine may show constipation. The passage of mucus is especially prominent.

A careful *differentiation* should be made between primary and secondary catarrh of the intestine.

Most important is the discovery of the cause of the catarrh.

Prognosis is more serious in infancy and old age, but depends largely upon the cause. In cases of long duration the

outlook becomes more unfavorable through the danger of atrophy or ulcer. In secondary catarrh of the intestine the prognosis depends chiefly upon the gravity of the primary disease.

Treatment: *Mild cases of intestinal catarrh* may require no treatment further than the removal of the cause, with abstinence from food for a short time and later a light diet, beginning possibly with diluted milk and gradually returning to the normal diet. Offending material must be removed from the intestine, best by calomel or castor oil internally and by cleansing enemata. Some prefer the biniodide of mercury. Abdominal pain and tenesmus may be relieved by warm or hot applications, or opium in the form of laudanum internally or the extract in suppositories. An excellent formula, given by Whittaker, is:

℞ Tincturæ opii, gtt. xl–lx;
Acidi hydrochlorici diluti, gtt. xl;
Aquæ camphoræ, ad ʒiv.
M. S. A teaspoonful to a tablespoonful every two to four hours.

Antiseptic and astringent remedies may be used internally and by enemata. Ewald recommends:

℞. Resorcin, (5.0) gr. lxxv;
Bismuthi salicylat.,
Tannigen, āā (15.0) ʒss;
Sacchari albi,
Sodii carbonatis, āā (7.5) ʒij.
M. ft. pulv.
S. Small even teaspoonful to be taken every two hours.

The intestine may be *irrigated* with a solution of nitrate of silver, with boric acid, tannic acid, or alum. Ewald prefers:

℞. Chloral, (3.0–5.0) gr. lxv–lxxv;
Acidi tannici, (1.5) gr. xxiv;
Lime water, ad (500.0) Oj.
M. S. One-quarter to one-third of this quantity is to be mixed with 12 ounces of warm water or thin starch-water, and of this 5 or 6 ounces or more may be injected into the bowel and should be retained as long as possible.

Carlsbad salt is one of the best laxatives.

Chronic catarrh of the intestine—treatment: Diet is most important. The drinking-water should be pure (boiled). In addition the treatment recommended for acute catarrh of the intestine may be indicated. Constipation frequently needs treatment. Sometimes a stay at one of the mineral springs may be necessary.

Catarrh of the intestine in infancy and early childhood: Food should be withdrawn for a time. During the interval the patient may be given boiled water, to which may be added a pellet of salt. Cases of chronic catarrh sometimes call for a change of diet. Lavage is often of value. Irritant material must be removed from the intestine, usually best by the administration of calomel or castor oil. Diarrhœa often is relieved by the administration of bismuth subnitrate, or tannalbin. More persistent cases may be relieved by a combination of ipecac and opium. Sometimes persistent vomiting is relieved by creosote.

ULCER OF THE INTESTINE.

Typhoid ulcer (see Typhoid Fever).

Tuberculous ulcer (see Tuberculosis).

Catarrhal and **follicular ulceration** have been treated of under Catarrh of the Intestine.

Round Duodenal Ulcer (Ulcus Duodeni Pepticum).

Ulcer of the duodenum is found in men more frequently than in women. It is believed that the gastric juice enters

the duodenum and causes corrosion when there has been a local circumscribed disturbance of circulation. Ulcer of the duodenum frequently appears after severe burns of the skin. Some attribute such cases to toxic material eliminated by the bile (Hunter); others believe it to be due to the liberation of the fibrin-ferment causing thrombosis of the duodenal veins.

Duodenal ulcer—symptomatology: Sometimes there are no symptoms. Some three hours after meals there may be *pain* in the region of the duodenum radiating toward the epigastrium and sacrum, sometimes very closely simulating gall-stones. The pain is not increased by food. Usually there is *diarrhœa*. More characteristic is *hemorrhage*, appearing as *melæna*, sometimes with hæmatemesis, very rarely as hæmatemesis alone. There are *tenderness* and *anorexia*. Sometimes *induration* may be detected.

Diagnosis: Duodenal ulcer should be *differentiated* from ulcer of the stomach, gall-stone colic, carcinoma of the duodenum, and the gastric crises of locomotor ataxia.

Ulcer of the stomach may be eliminated by the location of the circumscribed point of tenderness, the time of occurrence of the pain after taking food, the examination of the contents of the stomach, showing an absence of hyperchlorhydria, and an examination of the urine for peptone. The time of occurrence of pain and hemorrhage would speak against gall-stone colic. Carcinoma runs a shorter course, and frequently presents the symptoms of stenosis, and is accompanied by cachexia and more marked degradation of health, and often a tumor may be detected. The gastric crises of locomotor ataxia may be ruled out by the absence of symptoms on the part of the central nervous system, indicative of locomotor ataxia. The *prognosis* is not favorable.

Treatment: Special attention should be paid to the diet and all indiscretions avoided. Sometimes it is necessary to resort to rectal alimentation. Otherwise the treatment is symptomatic.

INTESTINAL HEMORRHAGE (Enterorrhagia).

Etiology: *Ulceration of the intestines* is the most common local cause of intestinal hemorrhage. Other conditions to

which hemorrhage may be due, are: inflammation of the intestinal mucosa, inflammation and ulceration following burns of the skin; intussusception; obstruction of the portal circulation; disease of the heart, lungs, bloodvessels, and liver; obstruction of the mesenteric arteries, and rupture of an aneurism into the intestine. Hardened fæces or foreign bodies passing through the intestine may cause slight hemorrhage. Hemorrhage may be caused by foreign bodies passed into the rectum by accident or design, as frequently occurs among the insane. The excessive use of purgatives or the ingestion of caustic or corrosive poisons may produce hemorrhage.

The infections may cause hemorrhage either through an action upon the mucosa of the intestine, as in the ulceration of typhoid fever, syphilis, and dysentery; or through an action upon the blood and vascular system, as in typhoid fever (before ulceration), yellow fever, plague, septicæmia, malaria, scurvy, purpura, hæmophilia, uræmia, and cholæmia. Hemorrhage from the intestine is sometimes an expression of vicarious menstruation. Hemorrhoids, ulcers, polypi, carcinomata, and tuberculosis intestinalis may be marked by hemorrhage.

Intestinal hemorrhage—symptomatology: There may be only *collapse*, without the passage of blood, sometimes without preceptible distention of the abdomen. Blood from the upper part of the intestines is sometimes regurgitated into the stomach and discharged by *hæmatemesis*. As a rule *blood appears in the stools*. The amount may be so small as to be detected only with the microscope, and sometimes blood may be discovered in this way before the appearance of gross hemorrhage (thirty-six hours before, Nothnagel). Hemorrhage high up in the intestinal canal may appear as black (tarry) stools. In hemorrhage from the colon, the passages may be covered or streaked with blood and mucus. When retained for a long time the blood may become inspissated.

Diagnosis: The stools should be carefully examined to detect the presence of blood. In doubtful cases an examination may be made with the microscope or spectroscope. Next the source of the hemorrhage should be determined, whether from the mouth, nose, pharynx, larynx, lungs, œsophagus, stomach, or intestine. Swallowing of blood is found

especially among malingerers, the new-born, and infants nursing from bleeding nipples. Examination of the anus and rectum may reveal hemorrhoids, ulcers, or polypi. Sudden collapse should awaken the suspicion of latent hemorrhage.

The **prognosis** depends largely upon the cause. A copious and persistent hemorrhage is always dangerous.

Intestinal hemorrhage—treatment: The foot of the bed should be raised. The diet may consist of milk, ice, and cold drinks. Cold applications—the ice-bag—should be placed over the abdomen. Opium may be given to restrain peristalsis. Liquor ferri persulphatis or pernitratis, tannic acid, or gallic acid, may be given internally. Ergotol, the aqueous extract of ergot, ergotin, or sclerotinic acid, may be injected subcutaneously. Astringent solutions, tannic acid, gr. j : ℥j of ice-water, or nitrate of silver, gr. ¼ : ℥j, may be injected into the bowel, when the hemorrhage comes from the rectum or lower part of the intestine. Any underlying condition should be properly treated. Thus malaria may call for quinin.

Collapse demands the use of the analeptics.

Lyman recommends—

℞. Moschi,
Pulv. camphoræ,
Pulv. capsici, *āā* gr. j.
M. Fiat pilula No. j.
Sig. Give one such pill every two to four hours.

Bad cases call for the use of camphor hypodermatically, best dissolved in olive oil or ether. Salt water infusion may rescue even desperate cases.

TYPHLITIS (Perityphlitis; Paratyphlitis).

Typhlitis (cæcitis) is a colitis limited to the wall of the cæcum. The chief *causes* are improper food and trauma, gallstones, enteroliths, foreign bodies, and the infections, typhoid fever, tuberculosis intestinalis, syphilis, dysentery, intestinal

diphtheria, carcinoma, and actinomycosis. When due to irritation from masses of fæces, the condition is known as *typhlitis stercoralis.*

Typhlitis—symptoms: The onset of symptoms is gradual. There is pain in the ileo-cæcal region, dull in character, sometimes paroxysmal, increased by pressure or movement, sometimes radiating to the umbilicus, right hypochondrium, or the epigastrium. The usual symptoms of dyspepsia, eructations, nausea, rarely vomiting of food or bile, are more or less marked. There are constipation, and usually some distention of the abdomen. A soft sausage-shaped tumor may be found in the right iliac fossa. The temperature usually is not very high, but frequently reaches 102° F. The urine, diminished in quantity and high colored, may contain small quantities of albumin and indican.

The **prognosis** of typhlitis is good.

In the **treatment** of simple typhlitis the bowels should be moved thoroughly with an enema of water or oil, combined if necessary with the internal use of castor oil. In simple typhlitis stercoralis the enema may be repeated, if necessary, with the administration of castor oil, calomel, or Carlsbad salt.

Perityphlitis, an inflammation of the peritoneal covering of the cæcum, and **paratyphlitis,** an inflammation of the peritoneum and connective tissue behind the cæcum (retrocæcal), as a rule, accompany appendicitis rather than typhlitis, although their relationship with typhlitis in some cases may not be denied.

"'Perityphlitis belongs to the surgeon,' has been until lately an assertion defended with emphasis by many surgeons, but which has never received the assent of the general practitioner, and never will. According to the experience of general practice, and the statistical results of Sahli, Renvers, Guttmann, Leyden, Fürbringer, Hollander, Rotter, and the majority of French physicians, from 90 to 91 per cent. of all cases of perityphlitis, taken in the widest sense, recover without any operation. It would, therefore, smack of insanity to subject every case of perityphlitis to the uncertainties of an operation" (Ewald).

APPENDICITIS.

Appendicitis: Inflammation of the vermiform appendix is usually not confined to the appendix, but extends to constitute a perityphlitis, or more correctly a *peri-appendicitis.*

Etiology: The infectious agent is usually the *bacillus coli communis*, sometimes associated with the streptococcus pyogenes or the staphylococcus pyogenes aureus or citreus or other bacteria, especially diplococci, usually as a secondary infection. Causes predisposing to such infection are: irritation due to arrest of the contents of the intestine, the continued retention of fæcal matter in the cæcum, bending or twisting of the appendix, and muscular relaxation of the wall of the appendix; excesses in eating, violent exercise soon after eating, and the ingestion of substances of an irritating (mechanical or chemical) character.

Bacteria grow readily in the mucus, sometimes mixed with fæcal matter, contained in the appendix. The appendix may be occluded or irritated by inspissated masses of fæcal matter, gall-stones, enteroliths, rarely foreign bodies, such as the eggs of ascarides, fish-bones or other small bones, bristles, hairs, and seeds. Sudden perforation may occur from necrosis of the wall of the appendix.

Appendicitis—symptomatology: Most cases occur between fifteen and thirty years of age, rarely before the third year, although the disease has been reported as early as the seventh week of life. Males are probably most frequently affected. There are often repeated *relapses or recurrences.*

A *simple catarrhal appendicitis* may show no symptoms. *Pain*, dull, boring or stabbing, may appear *in the ileo-cæcal region*, sometimes at the umbilicus or epigastrium, as repeated attacks of colic, which may occur in mild cases only when peristalsis is increased. A careful physical examination may reveal the swollen appendix.

More severe cases of appendicitis may show *dulness*, more or less marked, over the region of the appendix, and palpation may detect resistance resembling a *tumor* near the border of the right ilium. There are *pain*, *abdominal distention*, *dyspeptic symptoms*, sometimes vomiting of food, bile, medi-

cine, possibly stercoraceous vomiting, with *constipation* and some *fever*. The *urine* is scanty, high colored, gives the Burgundy-red reaction, and may contain indican.

In a *third class* of cases of appendicitis there is bacterial *invasion of the peritoneum*, sometimes but not always through perforation (necrosis, ulceration). There is violent abdominal pain after intestinal disturbances, trauma, the swallowing of some foreign body (bone, seed), sometimes without apparent cause. The pain later becomes localized in the region of the appendix. The temperature may rise above 102° F. *Hiccough* is a prominent symptom in many cases. Constipation is the rule; occasionally there is diarrhœa. *Movement causes pain.* There may be the symptoms of collapse. Physical examination may reveal a large perityphlitic accumulation.

In *chronic cases* the pus may burrow, to find exit in the most various ways.

Diagnosis: In the presence of typical symptoms the diagnosis is easy. Of most importance, as a rule, are the previous history and the presence of a tumor or dulness in the region of the appendix. Tenderness may be elicited at *McBurney's point.*

The **differential diagnosis** calls especially for the separation of renal colic, hepatic colic, and in chronic cases cancer and tuberculous peritonitis. Often appendicitis is tubercular. The inflammation may not be confined to the usual region of the appendix, so that it may be necessary to rule out perinephritic inflammation, hæmatocele, salpingitis, pyosalpinx, cholelithiasis, or disease of the liver; or to recognize an appendicitis occurring in a hernial sac in the scrotum or elsewhere. Confusing symptoms may sometimes be caused by constipation from opium, invagination, or strangulation. Sometimes a positive diagnosis can be made only upon exploratory incision.

Appendicitis—prognosis: When appeal is to be made to surgery, the earlier such treatment is instituted the better will be the prognosis. Suppurative peritonitis gives a bad outlook. The occurrence of complications increases the gravity of the case. The prognosis is unfavorable when the peritonitis

becomes general, or in the presence of a suppurative pleurisy, pylephlebitis, or abscess of the liver.

Prophylaxis: The bowels should be regulated. Especially must constipation be remedied. Food is to be avoided that would cause constipation or leave a large residue in the bowel. The meals should be eaten slowly and thoroughly masticated. The patient should rest for a while after meals. Indigestible substances, such as the seed of fruit, should not be swallowed. The danger of trauma, as far as possible, must be avoided.

Appendicitis—treatment: During the attack the patient must observe absolute *rest in bed*. The *diet* should be light; best nothing but boiled water and cracked ice for the first twenty-four to thirty-six hours. Later milk may be given, cold or lukewarm; then small quantities of oatmeal, farina soup, or bouillon made from white meat. The return to the normal diet should be very gradual.

Pain calls for the use of opium, with applications of cold (cold compresses, ice-bag), or of heat (hot compresses, poultices), and infusions of chamomile, valerian, etc.

Constipation, even when obstinate, may be allowed to run several days during the acute stage of an attack without treatment. The bowels may then be gently moved by small enemata carefully given. Should these fail, high injections may be given of water containing castor oil, made into an emulsion with the yolk of an egg. When necessary, purgatives may be administered internally, best castor oil; later calomel or Carlsbad salt may be given.

Operation may be demanded by perforation, suppuration, recurrence of attacks with increasing severity, when the operation should be made between attacks, and in cases of chronic appendicitis with indefinite and obscure symptoms. It is not infrequent to find an apparently healthy appendix at operations in cases in which the clinical symptoms have been those of appendicitis (Ramm).

INTESTINAL OBSTRUCTION (**Ileus**; **Intestinal Occlusion**).

Etiology: Obstruction of the intestine may be caused by abnormal conditions of the intestine, of the intestinal con-

tents, or by compression from tumors, misplaced organs, etc. Chronic or habitual constipation may be present even when there is the history of daily stools. The condition may be due to atony of the bowel or a weakening of the muscle-fibres of the intestine through inefficient efforts to overcome some chronic obstruction of the bowel; irregular habits of defecation; leaving too much or too little residue to pass through the intestine, or a diet without variety, such as an exclusive meat-diet, or food containing too little water, or the loss of water through the skin (profuse perspiration), lungs, or kidneys; abuse of purgatives, fatiguing the muscular fibres; change from an active outdoor to an indoor sedentary life; mechanical pressure (pregnancy); preoccupation of the mind by business or domestic cares, melancholia, insanity.

Intussusception, or *intestinal invagination*, may occur physiologically during the so-called death-agony, and possibly at other times. Intussusception occurs pathologically most frequently in children, half the cases before the tenth year; in males more frequently than in females. The condition occurs, in the order of frequency, as ileo-cæcal invagination, enteric invagination, and colic invagination. Invagination of the duodenum is rare.

Internal strangulation usually affects the small intestine, the lower part of the ileum, and is most frequent under forty.

Stricture may result from cicatrization following ulceration (dysentery, less frequently typhoid fever, tuberculosis). More often stricture is due to syphilis or carcinoma and may be recognized by rectal examination.

Obturation of the intestine may occur from the lodgment of gall-stones, more rarely from enteroliths, as a rule having as a nucleus some foreign body. Calculi may be caused by the continued medicinal use of mineral substances, such as calcium carbonate, calcined magnesia, or magnesium carbonate.

Volvulus, a turning of the intestine around its mesentery or upon its own axis, occurs most frequently after forty, usually at the sigmoid flexure. Volvulus is caused as a rule by chronic constipation, sometimes by chronic peritonitis, and may be, rarely, congenital. Twisting of the intestine has

been ascribed to violent irregular movements of the intestine, jumping, trauma, and the use of high injections. In such cases peritonitis may occur early, but perforation is rare. Without operation death usually occurs in two to six days. One case is reported to have lived twenty days (Treves). Twisting of the intestine upon its own axis occurs only in the cæcum and ascending colon. Volvulus of the ileum or jejunum is rare.

Collapse may supervene suddenly. There is violent pain, often intermittent, with vomiting, slight in quantity but fæcal in character. The convolution above the volvulus is distended and fixed. It is impossible to introduce water or air through the rectum past the obstruction. There is early tenesmus. The urine does not contain indican. There may be only half a turn of the bowel, constituting an incomplete volvulus.

In addition to the *causes already enumerated*, obstruction of the intestine may be caused by compression from a tumor or from some other organ: displaced uterus, dislocated spleen, floating kidney.

Cases in which no other cause can be found are supposed to be due to a circumscribed intestinal paralysis, so-called cases of **paralytic ileus**. The condition may be only an arrested peristalsis or an actual paralysis, and may be due to trauma, appearing sometimes after the reduction of a hernia; inflammation, peritonitis, or to other causes which may not be easily discovered.

Symptomatology: The onset of the symptoms of *ileus* may be gradual or sudden. Where the obstruction is complete, the *constipation* may amount to *obstipation*. Usually there is *pain*, which may be present in every grade of severity, and either intermittent or constant. There are *tormina and tenesmus*. The pain comes on earlier and is more marked when the small intestine is involved. The pain is not always referred to the point of obstruction. There are singultus; eructations; later *vomiting*, which is at first, or soon becomes, stercoraceous. The abdomen is more or less distended and tender. Percussion reveals *meteorism* in cases of acute ob-

struction, which is absent in the occlusion from constipation. The *urine* is diminished in quantity, of high specific gravity, and contains urates. *Indicanuria* appears early in obstruction of the small intestine, and late or not at all in obstruction of the large intestine, and is small in amount when the obstruction is in the upper part of the small intestine.

Complications: The chief complications are peritonitis, perforation, and fistula.

Ileus—diagnosis: Usually easy. The condition must be differentiated especially from general peritonitis, acute typhlitis and perityphlitis, appendicitis, and coprostasis; occasionally from Asiatic cholera, strangulation of a floating kidney, renal calculus, lead-colic, displacement of the uterus, and ovarian tumors.

It is often more difficult to recognize and locate the *cause* of the obstruction. When the obstruction (tumor, carcinoma, syphilis) is in the *lower part of the large intestine*, it may be detected by rectal examination. Obstruction in this part of the bowel will permit the introduction of only a small quantity of water or air per rectum. Sometimes only small enemata may be used in patients who are nervous or neurasthenic, or who are not accustomed to such injections, even when there is no obstruction. Cases of obstruction in the lower part of the large intestine show an absence of indicanuria. In such cases the symptoms of ileus come on later, there is considerable meteorism, and there is not so great collapse.

When the obstruction is in the *jejunum* or *upper part of the ileum*, there is early vomiting of bile (not stercoraceous), sudden collapse, and absence of general distention of the abdomen. Indican is absent from the urine, the same as when the obstruction is in the rectum. The location and character of the pain may be deceptive, but often will give a clue to the location of the obstruction.

Prognosis: Depends upon the cause, whether the occlusion is complete, and the condition of the patient. In severe cases the prognosis is bad, and even in cases less severe the mortality is high.

Ileus—treatment: An uncomplicated fæcal obstruction

may be relieved by purgatives. In other cases all purgatives may do harm. Intestinal irrigation not only cleanses the bowel, but may relieve strangulation before the formation of adhesions. Rectal injections often suffice to reduce a volvulus or intussusception. The introduction of air into the bowel increases peristalsis and causes distention of the intestine, but does not remove the contents so well as when water is used. Repeated lavage of the stomach may relieve the symptoms by lessening the tension on the intestine, and has been reported to effect a cure by exciting reflex movements in the intestine, in cases of an incarcerated convolution, intussusception or volvulus.

Opium in suppositories, or morphine subcutaneously, may be given to overcome peristalsis and pain. While administering opiates the physician should not underrate the gravity of the case.

Intestinal convolutions distended by gas may be relieved by puncture, under strict asepsis; but, especially in cases of intestinal paralysis, *there is danger* of some of the contents of the intestine getting into the peritoneal cavity through the puncture and causing peritonitis. Sometimes the paralysis caused by the passage of foreign bodies may be relieved by massage and electricity. Excessive thirst may be relieved by rectal injections of water and permitting the patient to drink all the water he wants, with lavage of the stomach. Infusion of salt solution may be called for.

Many cases can be relieved only by surgery. When an operation is decided upon, the earlier it is done the less will be the danger. A primary enterostomy or colotomy should be made, if the patient is not in condition to undergo a more prolonged operation, and a secondary operation may follow when the patient is in better condition.

ENTEROPTOSIS.

The term, **enteroptosis**, is used to indicate a descent or displacement of any of the abdominal viscera, as well as a downward displacement of the intestine. A descent of the transverse colon, *coloptosis*, is the most frequent intestinal displace-

ment. Prominent causes are constipation and violent exertion. There are frequently emaciation and indigestion. Enteroptosis is most frequent in cases of anæmia and neurasthenia, which condition it aggravates.

The **diagnosis** may be made by percussion of the bowel after it is distended with air.

Treatment calls for the relief of constipation, attention to the general health, especially the treatment of anæmia and neurasthenia, and the support of the abdomen by a well-fitting bandage or supporter.

HEMORRHOIDS (Piles).

Etiology: Hemorrhoids occur most frequently from thirty to fifty years of age. Hemorrhoids may be caused by anything which interferes with the return of blood from the hemorrhoidal veins. Prominent causes are constipation, obstruction of the portal circulation (cirrhosis of the liver), chronic proctitis, stricture of the rectum, pelvic or abdominal growths or tumors, pregnancy, enlargement of the prostate, and diseases of the heart and lungs that may cause congestion of the hemorrhoidal veins by interference with the circulation of the blood.

Symptomatology: The patient feels as if a foreign body were in the rectum, which causes burning and smarting, often painful defecation, sometimes painful micturition. In cases of *internal hemorrhoids* there may be a mucous or muco-purulent discharge, sometimes mixed with blood.

Diagnosis: External hemorrhoids may be revealed by inspection; internal hemorrhoids, by inspection through the rectal speculum or by digital examination.

Prognosis: As a rule good as far as life is concerned. Rarely the hemorrhoids may become strangulated or gangrenous. Relapses are frequent, except when thorough surgical treatment is resorted to.

Hemorrhoids—treatment: As far as possible the cause should be removed. Constipation should be relieved; exercise should be prescribed for the indolent and those engaged in sedentary occupations. The patient should abstain from

irritating food and drink, such as strong spices, alcoholic drinks, strong coffee and tea, acid articles (pickles), and food that contains much material that will be left as a residue in the intestine. Upholstered chairs and feather beds are to be avoided. After defecation the anus should be sponged, best with a weak solution of carbolic acid or some antiseptic, and dried with lint.

For symptoms of irritation, especially after excoriations, the application of an ointment of vaseline, lanoline, or cocoa-butter, containing morphine, extract of belladonna, or cocaine, may give relief. The hemorrhoids may be touched with a 2 per cent. solution of cocaine, or a 1 per cent. solution of nitrate of silver. Inflammation may be relieved by ice-water or ice bags, poultices, or hot baths.

Pain may be relieved by ointments (unguentum gallæ cum opii), or suppositories of opium or morphine and atropine. Hemorrhage may call for the administration of calomel and bicarbonate of sodium, or the local application of ice plugs, tampons, or injections of hot water, and astringent solutions, tannic acid, alum, acetate of lead, or nitrate of silver. Prolapse and strangulation or very great discomfort call for the intervention of surgery. Injections of carbolic acid, 1 : 3 of glycerin, gtt. v into each pile, may be repeated at intervals of a few days. These injections may be preceded by injections of a 1 per cent. solution of cocaine or the Schleich fluid. Care should be taken not to make the injection into the cellular tissue, because of the danger of abscess.

INTESTINAL NEOPLASMS.

Carcinoma is the most frequent intestinal neoplasm, but does not occur as often as carcinoma of the stomach. Most cases occur from forty to sixty years of age. Only one-seventh of cases occur before thirty (Maydl). From an examination of the statistics regarding the location of carcinoma of the intestine, Ewald found the rectum involved 874 times, the large intestine 148 times (the transverse colon 12 times), the cæcum including the appendix 64 times, the ileum 26 times, the duodenum 19 times, and the jejunum 17 times.

Symptoms: Often carcinoma may exist some time before symptoms are noticed. The most characteristic symptoms are the peculiar *cachexia*, the presence of a *tumor*, *malnutrition*, and *obstruction of the bowel*. The obstruction of the bowel may disappear under treatment to reappear again later. The condition is chronic. Other symptoms will depend upon the location of the carcinoma. Sometimes a tumor may not be detected.

Diagnosis: In the presence of a complete array of symptoms, especially cachexia, tumor, and intestinal obstruction, the diagnosis is easy. Sometimes a piece of the neoplasm may be removed, through a rectal speculum or endoscope, or through an incision, and examined microscopically, to make the diagnosis absolute. Intestinal carcinoma should be *differentiated* from tuberculosis, syphilis, dysentery and typhoid fever, carcinoma of the pylorus or of the gall-bladder, pancreas or omentum, echinococcus of the omentum, retroperitoneal neoplasms, neoplasms of the uterus and its adnexa, intestinal concretions (gall-stones, fæces), appendicitis, and tumors of the kidney or spleen.

The **prognosis** is unfavorable. Such patients may live for a number of years. The duration of life is usually longest when the carcinoma is situated in the rectum.

The **treatment** is surgical, and should be resorted to as soon as a diagnosis is made. The results are not so good after the formation of metastases or adhesions. Often marked improvement follows colotomy.

Sarcoma and **lymphosarcoma** of the intestine are rare. The most common site is the small intestine. There is a marked tendency to metastases, and the course is more rapid than in carcinoma of the intestine.

Among the **benign neoplasms** of the intestine are adenomata, fibromata, lipomata, papillomata, angiomata, myomata, fibromyomata, myxomata, and fibro-myxomata.

Most important are the *intestinal polypi*, tumors having a pedicle, which may cause intestinal obstruction. In the small intestine they may cause invagination. Rectal polypi may be

detected by digital examination. All such tumors may cause diarrhœa, with the discharge of mucus, pus, or blood, or intestinal obstruction and hemorrhage.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Etiology: Acute inflammation of the peritoneum is caused chiefly by bacterial infection, most frequently by the bacillus coli communis, streptococcus pyogenes, staphylococcus pyogenes aureus, citreus and albus, sometimes by the micrococcus pneumoniæ crouposæ, bacillus lactis aërogenes, bacillus typho-abdominalis, and the proteus vulgaris. These bacteria may gain access to the peritoneum through solutions of continuity (perforating ulcers, wounds) of the stomach, intestine, or abdominal wall, or through the intact walls of the stomach, intestine, or other abdominal or pelvic organ covered by peritoneum, when injured (trauma); or through extension of inflammation of abdominal or pelvic organs (appendicitis, genito-urinary diseases).

Peritonitis, due to the micrococcus pneumoniæ crouposæ, is found most frequently in girls. When localized, the process is found in the pelvis. Boulay found the micro-organism in the uterus, which would seem to indicate that the channel of infection of the peritoneum is from the uterus, through the lymphatics or tubes (F. Brun).

Pernice produced peritonitis *experimentally*, in rabbits and guinea-pigs, by injecting chemical substances (concentrated mineral acids, acetic acid, phenol, nitrate of silver) into the peritoneal cavity. Sternberg failed to produce peritonitis in rabbits by the introduction of sterilized powdered glass into the abdominal cavity.

Other causes of acute peritonitis are general septicæmia, miliary tuberculosis, malaria, dysentery, nephritis and suppurative inguinal adenitis (Fitz).

Symptomatology: The principal symptoms of acute peritonitis are *abdominal pain and tenderness*, *constipation* (sometimes diarrhœa), *nausea*, often *vomiting*. Usually the *tem-*

perature is high and the *facial expression anxious*. *Thirst* is often distressing. There is *singultus*. The *abdomen is at first retracted, later distended and tympanitic*. The abdominal muscles are firmly contracted. *Respiration* becomes largely or altogether costal. The *pulse* is rapid, small, and wiry. Frequently there is *retention of urine*. Sometimes there is collapse.

Pain and tenderness may be absent, especially in septic (puerperal) peritonitis. Sometimes the peritonitis is limited to a part of the abdominal or pelvic cavity to which the symptoms are confined, constituting a localized peritonitis.

Complications and **sequelæ of peritonitis**: There may be tympanites, from paralysis of the bowel; obstinate vomiting, probably reflex through the pneumogastric nerve; retention of urine, through extension of inflammation from the peritoneal covering to the muscular wall of the bladder; bronchitis, pneumonia, pleurisy, or strangulation of the bowel.

Diagnosis: The abdominal pain and tenderness, board-like rigidity of the abdominal muscles, and constipation, form a characteristic group of symptoms of acute peritonitis. More difficult to diagnosticate are the cases in which abdominal pain and tenderness may be absent. Acute peritonitis should be differentiated from obstruction of the bowel, acute hemorrhagic pancreatitis, hysterical peritonitis, gall-stone and renal colic, and subphrenic abscess.

The **prognosis** should be guarded.

Acute peritonitis—treatment: If possible, the cause should be found and removed, if necessary by the intervention of surgery.

The medical treatment consists in the relief of pain and vomiting by the administration of opium internally or morphine hypodermatically. Turpentine stupes and mustard plasters may be applied to the abdomen. Some cases may be relieved by high rectal injections, which may contain turpentine and milk of asafetida. Alcohol, best in the form of wine, whiskey, or brandy, is sometimes useful.

In the absence of vomiting, food may be given by the mouth; but usually it is best to give the stomach a rest. The patient may be sustained for a few days by nutritive enemata.

The judicious use of saline purgatives is often of very great value.

Sometimes the cause may be found and removed by an exploratory incision. As in so many other conditions, when an operation is demanded the earlier it is performed the better are the chances for recovery.

TUBERCULAR PERITONITIS.

Definition: An infection of the peritoneum by the tubercle bacillus.

Etiology: The *tubercle bacillus* infects the peritoneum most frequently by direct extension from the intestine, next in frequency from the female genital organs. The disease occurs especially in early adolescence, but infancy and old age are not exempt.

Symptomatology (see Symptomatology of Acute Peritonitis): Usually the symptoms of tubercular peritonitis are not so acute, and the course is more chronic. *Abdominal distention and rigidity*, *emaciation*, and the streptococcus *fever-curve* are present. There may be symptoms on the part of the organs primarily affected, especially the lungs, intestine, and genital organs.

The temperature may be subnormal for days at a time (Osler, Musser). Sometimes tubercular nodules are recognized by palpation, especially upon vaginal or rectal examination (Heger). There may or may not be ascites.

The disease may continue for months or years, or early show the symptoms of miliary tuberculosis or acute peritonitis.

Diagnosis: Sometimes tubercle bacilli may be found in the ascitic fluid. Often a test-injection of tuberculin will clear up a doubtful case.

Differential diagnosis has to do chiefly with chronic peritonitis (not tubercular) and cirrhosis of the liver.

Prognosis: Some cases may be rescued only by operation and the exposure of the peritoneum to the air. Cures have resulted from puncture and aspiration, and spontaneously. Many cases terminate favorably under medical treatment.

Tubercular peritonitis—treatment: Many cases may be res-

cued by the use of tuberculin R, climatotherapy, cod-liver oil, and massage. Laparotomy, with the removal of the primary focus in the intestine or female genital organs (Fallopian tube), sometimes gives the best results. It may be necessary to make repeated laparotomies. Aspiration and the injection of air and other substances into the peritoneal cavity have been unsatisfactory.

CHRONIC PERITONITIS.

The most frequent form of chronic peritonitis is **tubercular peritonitis**, which has already been discussed.

The **etiology** of chronic peritonitis, *aside from tubercular peritonitis*, is obscure. Exposure to cold has been invoked as a cause, as have also the causes of acute peritonitis in the presence of sufficient resistance to prevent an acute attack. Localized chronic peritonitis is most frequently due to subacute pelvic inflammations in which the irritation is not sufficient to produce an acute peritonitis. Sometimes cases may be caused by repeated paracentesis abdominalis (tapping).

Symptomatology and diagnosis: The symptoms resemble those of acute peritonitis, but are, as a rule, less intense. The distinction between tubercular peritonitis and peritoneal neoplasms is sometimes impossible. Most important is the discovery of the cause.

The **prognosis** is usually good. The greatest danger arises from complications, especially strangulation of the bowel and pressure upon important abdominal organs.

The **treatment** is the treatment of peritonitis in general (see Treatment of Acute Peritonitis). Ascites may be relieved by paracentesis.

PERITONEAL NEOPLASMS.

Carcinoma rarely occurs in the peritoneum primarily. Secondary involvement of the peritoneum is not uncommon. The *treatment* is purely surgical

Hydatid cysts occur most frequently in the abdominal organs, but may occur in the peritoneum. *Diagnosis* often

rests upon exploratory incision. Aspiration is dangerous. The *treatment* is surgical. It is better to remove the cyst without puncture.

Other tumors of the peritoneum are sarcoma, lipoma, and fibroma. More rare are myxoma, endothelioma, hæmangioma and chylangioma, papillary cystoma, dermoid and teratoid cysts.

The **treatment** of neoplasms, when active treatment is demanded, belongs to surgery. Otherwise the treatment is largely symptomatic.

DISEASES OF THE LIVER.

ICTERUS (Jaundice).

Icterus: *Jaundice* is a yellowish color of the tissues and fluids of the body.

Etiology: Icterus may be caused by anything that interferes with the flow of bile. Such interference with the flow of bile may be caused by occlusion of the bile-ducts through swelling of the mucous membrane of the ducts, the presence of inspissated mucus, the impaction of a gall-stone, or by the pressure that may be caused by cancer of the stomach, liver, pancreas, omentum; or by sarcoma; or by the cicatrization of an ulcer in the duodenum or bile-ducts, or by abdominal tumors, vertebral caries, aneurism, etc.

Symptomatology: The skin and mucous membranes present a change in *color varying from a light yellowish tinge to a dark yellow. Itching*, pruritus, is usually noticed after jaundice has existed for some time, but may even precede the appearance of the change in color. There may also be various cutaneous eruptions.

There is *interference with intestinal digestion and absorption. Fat appears in the stools* in large amounts. The *fæces are pale*, from the presence of fat and the absence of bile. The absence of the normal stimulus of the bile upon the intestine results in a lessened peristalsis and consequent *constipation.* The absence of the antiseptic action of the bile permits abnormal *fermentation and putrefaction* of the fæces and causes *meteorism.*

The *secretions* and *excretions*, urine, perspiration, milk, sometimes the sputum, may contain bile-pigments.

The presence of bile in the blood, cholæmia, causes *auto-intoxication*. The *pulse*-rate is lowered (bradycardia), but in long-continued cases of grave icterus the pulse may be normal or quickened (tachycardia). Sometimes the pulse is irregular. The *number of red blood-corpuscles is reduced*, especially when icterus has existed for some time.

The *temperature* is subnormal, unless elevated by some cause other than the icterus. Long-standing cases may show *hemorrhages* from the mucous membranes, especially epistaxis, gastrorrhagia, and enterorrhagia.

The chief *nervous symptoms* that may occur are delirium, coma, convulsions, muscular tremors, and paralysis of the sphincters. Often the patient is in a typhoid condition.

Sometimes there is a bitter taste in the mouth, and often the tongue is coated.

Diagnosis: In light cases the change of color may be observed in the conjunctivæ as a slight yellowish tinge. In marked cases the yellow color of the skin is obtrusive. Bile-pigments may be detected in the urine. For this purpose various tests have been proposed.

Gmelin's test consists in floating a layer of urine upon nitrosonitric acid, when at the point of contact there will be observed a distinct green color, representing the oxidation of bilirubin into biliverdin. Other colors may be present, but are not indicative of the presence of bile-pigment. Rosenbach has modified this test by filtering the urine and placing upon the filter-paper thus used a drop of fuming nitric acid, when the color will appear as above. V. Jaksch remarks that the only caution to be observed in this modification is that the filter-paper must be pure and white. Pure white blotting-paper may be used and is quite satisfactory. The blotting-paper is soaked with the urine and a drop of the acid added. The green color forms a distinct ring.

A simple test suggested by Marechal consists in adding to the urine a few drops of the tincture of iodine. An emerald-green color indicates the presence of bile.

The bile-acids may be detected by *Pettenkofer's test*, best as

modified by Strassburger. A piece of cane-sugar is dissolved in the urine, with which a piece of blotting-paper is then moistened. After drying, this is touched with pure concentrated sulphuric acid. A positive reaction consists in the development, at the point of contact, of a carmine-violet-purple color.

Icterus should be *differentiated* from Addison's disease, cachexia, and a normal yellow color of the skin.

The **prognosis** is that of the disease causing the icterus.

Icterus—treatment: As far as possible, treatment should address the underlying disease, usually catarrh of the bile-ducts, gall-stones, syphilis, malaria, or carcinoma.

The *diet* should consist of articles that will not irritate the liver: milk, soft-boiled eggs, bouillon, meat soups, and thoroughly cooked fruit. Alcoholic beverages and foods containing fat should be withheld.

In the way of *medicines*, the salicylates, salol, phosphate of sodium, benzoate of sodium, benzoic acid, calomel, and hydrochloric acid are useful. In some cases hepatic stimulants may be used, especially podophyllin, jalap, and colocynth.

Among the *mineral waters* in common use are Carlsbad, Vichy, Ems, Selters, Hathorn, Saratoga, and for some cases Marienbad, Kissengen, and Homburg.

The salicylates not only act antiseptically, but also increase and liquefy the flow of bile. Inspissated ox-gall may be given internally as a purgative and antiseptic, or fresh ox-gall may be used in enemata. Large enemata of hot water (Bouchard) have been recommended in cases of threatened uræmia and may be of value in cholæmia.

GALL-STONES (Cholelithiasis; Biliary Lithiasis).

The **concretions** found in the *gall-bladder* and *biliary passages* vary in size, shape, color, and composition. The chief constituents are cholesterin, bile-pigment (bilirubin, associated with biliverdin, bilicyanin, and bilifuchsin), and salts of lime and magnesia. The nucleus is sometimes a foreign body (bacillus coli communis; mercury). The stones are frequently laminated. Usually they are faceted.

Etiology: About two-thirds of the cases occur in women. Cases are more frequently encountered after forty, rarely under twenty, although gall-stones have been found in the new-born. Conditions which interfere with the flow of bile, sedentary habits, old age, bad hygiene, diabetes, carcinoma of the stomach and liver, are predisposing causes.

Morgagni has observed that gall-stones and kidney-stones are frequently found in the same individual. Patients with gall-stones are often subjects of obesity, rheumatism, gout, lithæmia, and atheroma.

Catarrhal inflammation of the bile-ducts may be a predisposing cause, and the cholesterin may be precipitated on the necrosed epithelium. It is possible that typhoid fever may play a rôle in etiology, and many times cases have been attributed to the drinking-water. Gall-stones have been observed more frequently in some countries (Hanover, Sweden, Hungary) than in others (Holland, Finland).

Symptomatology: Calculi in the gall-bladder may produce no symptoms. Post-mortems show gall-stones in about one-tenth of all cases, most frequently in the female sex. The most obtrusive symptom is *pain*, gall-stone colic. An overdistended gall-bladder may be painful. The attack of gall-stone colic begins with a feeling of discomfort, which gradually increases to absolute, often excruciating, pain, in the right hypochondrium or epigastrium. *There is interference with digestion, sometimes vomiting.* Often the patient complains of pain at the angle or inner margin of the scapula. There are *obstipation* and *tympanites.* There is a slight rise of *temperature,* 99°–99.5° F. *Usually there is no icterus.* As a rule, gall-stones do not cause colic, except when there is impaction of the stone in the cystic duct. When the gall-bladder is enlarged it may be felt as a tumor.

Diagnosis: Gall-stones should be differentiated especially from ulcer of the duodenum and ulceration or malignant disease of the pylorus, hepatic carcinoma, and obstructions of the bile-ducts from other causes.

Prognosis: The duration of an attack of biliary colic due to gall-stones is usually three to six days. Frequent recurrence is the rule. The advances in surgery have made the outlook

much more favorable. Surgery may give a mortality not exceeding 5 per cent., except in cases of jaundice and malignant disease. The prognosis of malignant cases is bad.

Prophylaxis: High living, rich diet (brains, yolks of eggs), and stimulants (ale, porter, wine) should be avoided. The use of tight-fitting waistbands and corsets should be abandoned. Bicycling and horseback-riding are forms of exercise highly recommended. In the way of medicinal prophylactics, salicylic acid, cholate of sodium, the sulphate and phosphate of sodium an hour before meals, deserve mention.

Gall-stones—treatment: Pure olive oil, a wineglassful at bedtime every night, may do good in some cases. If necessary, the oil may be substituted by glycerin. Sometimes the salicylate of sodium may be of value. Sulphuric ether and chloroform have been used; they probably act as antispasmodics rather than as solvents of the calculi. Durande used a mixture composed of three parts of sulphuric ether and two parts of oil of turpentine, a drachm each morning. Graham suggests that gtt. xx three times a day would be better tolerated by the stomach. Large quantities of water, hot water with or without bicarbonate of sodium, ʒj–ij : Oj, or mineral water, especially Carlsbad and Vichy, are often beneficial. Sodium sulphate has been recommended, ʒj-ij, taken before breakfast in a bitter infusion, with sodium bicarbonate, gr. xx-xxx at bedtime (Harley). For the indigestion caused by the cutting off of the bile from the intestine, ox-gall, cholate of sodium, acetic acid, and citric acid have been recommended.

Pain may call for the hypodermatic use of morphine, to which belladonna or atropine may be added. Sometimes relief is obtained by the local application of heat, or better by the hot bath. Severe pain may demand chloroform or ether.

Surgery offers the most hope for absolute cure. The operation to be selected will depend upon the conditions present in the individual case. Among the operations commonly performed for gall-stones are: *cholecystostomy*, in which the gall-bladder is opened, the stones removed, and the gall-bladder stitched to the abdominal wall; *cholecystectomy*, complete removal of the gall-bladder; *cholecystenterostomy*, in which a direct communication is established between the gall-bladder

and intestine; *choledochotomy*, *choledocholithotomy*, in which the common duct is opened for the removal of a gall-stone. Operations less frequently performed are *cholecystendysis*, removal of stones from the gall-bladder by incision, closure of the incision by suture, and return of the gall-bladder to the abdominal cavity; *choledocholithotripsy*, in which the gall-stone is crushed in the common duct; and puncture of the gall-bladder. Calculi have been forced from the common bile-duct by external pressure, a procedure not devoid of danger, especially of rupture.

ACTIVE HYPERÆMIA.

Hyperæmia of the liver occurs physiologically during digestion, and may become excessive after indulgence in food of an irritating nature (alcohol, spices), or which may cause intestinal fermentation. Persistence in such indiscretions may cause a permanent pathologic hyperæmia of the liver.

Hyperæmia of the liver is common in the infections, especially typhoid fever, malaria, and dysentery. Gout is a frequent cause. Some cases are due to syphilis.

Among the **toxic causes** are alcohol, carbonic oxide, mercury, carbolic acid, phosphorus, arsenic, nicotine, etc.

Tropical hyperæmia of the liver is probably largely due to infection. Some cases are attributed to nervous causes acting through the vaso-motor nerves.

Hyperæmia of the liver—symptomatology: The onset may be gradual, with a feeling of tension in the epigastric region and dyspeptic symptoms. Usually the symptoms begin rather suddenly, with a slight *chill* and *fever* followed by *pain* and a *feeling of tension* in the *region of the liver*, *radiating to the right shoulder*. *Dyspeptic symptoms* appear early, sometimes before there is pain, and become pronounced. There are *nausea*, *vomiting*, and *diarrhœa*. Slight *icterus* appears in two or three days, and may become severe. The *fæces* are colored (pleochromatic). The *urine* is reduced in quantity, with high specific gravity, contains bilirubin or urobilin, and an increased amount of urea, 40–50 grammes in the twenty-four hours. Severe cases may show some enlargement of the

spleen. More prominent is the *enlargement of the liver*, palpation of which is usually painful.

Diagnosis: The chief diagnostic points are the severity of the symptoms, the elevation of temperature, the general condition of the patient, and the course of the disease. Most important is the determination of the cause, and the separation of active hyperæmia from cirrhosis of the liver and abscess of the liver.

The **prognosis** is usually good when the cause can be removed.

Hyperæmia of the liver—treatment: Should first address the cause. The diet must be regulated, alcohol withheld, and gout, malaria, or other infectious disease properly treated. Usually a milk-diet is best. An initial dose of calomel may be followed by the use of saline purgatives or mineral waters. Carlsbad and Vichy are recommended as watering-places for chronic cases. Cold is applied to the region of the liver, in the form of an ice-bag or cloths wrung out of ice-water. Intestinal antiseptics are used.

PASSIVE HYPERÆMIA.

Passive hyperæmia of the liver is caused chiefly by obstruction of the general circulation, which may be due to valvular disease of the heart, myocarditis, pericarditis, arteriosclerosis, aneurism, marasmus, pleurisy, empyema, asthma, bronchitis, emphysema, or pneumonia. Alcoholism and the infections, especially malaria, may account for these causes producing passive hyperæmia of the liver in some individuals and not in others. Some cases are due to obstruction in the hepatic veins.

Symptomatology: There is *enlargement of the liver*, sometimes with persistent *pain*, which may be increased by palpation. There may be *anorexia*, *vomiting*, *eructations*, *and constipation alternating with diarrhœa*. Obstruction of the hepatic circulation may cause *hemorrhoids and hemorrhage from the intestine*. Often there are *ascites* and the formation of the *caput Medusæ*. The *urine* is diminished in quantity, high colored, of high specific gravity, and usually contains urobilin and uroerythrin,

and in marked cases of jaundice pure bile-pigment. Urea may be diminished or increased with a diminution or increase in the volume of urine voided. Uric acid and the chlorides are decreased; the phosphates are increased. There may be glycosuria, sometimes albuminuria.

The **diagnosis**, in the presence of enlargement of the liver, is easy when an etiological factor, such as a pulmonary or cardiac lesion, is obtrusive.

The **prognosis** depends largely upon the cause, the condition of the patient, and the severity of the case.

Treatment: A failing heart must be stimulated. A milk-diet should be adopted. In the way of medicines, calomel, the saline purgatives, and mineral waters are most important. Calomel and diuretin may be used as diuretics.

Cases which resist other treatment may be subjected to paracentesis, repeated as often as necessary, from which very good results may be obtained.

ABSCESS OF THE LIVER (Suppurative Hepatitis).

Etiology: The principal causes are pyæmia, inflammation of the bile-ducts, dysentery, appendicitis, suppurating glands, ulceration from gall-stones, suppurative pylephlebitis, umbilical phlebitis (in the new-born), tuberculosis, foreign bodies, and parasites.

Tropical abscess in some cases depends upon the presence of the amœba coli, but in many cases the amœba may not be found. The pus-producers, especially the staphylococcus pyogenes aureus and staphylococcus pyogenes albus, are frequently present. It is supposed that alcohol, through the production of acute hyperæmia of the liver, is a predisposing cause. Some cases may be due to toxins. The absence of micro-organisms may be due to their destruction by the liver.

Abscess of liver—symptomatology: *Symptoms may be absent or indefinite*, or overshadowed by sepsis or trauma.

The *acute form*, found especially in hot countries, begins with *malaise*, *chills*, and *fever*, sometimes remittent or intermittent, higher in the evening and at night. The region of the *liver becomes painful*, especially upon pressure, with a feel-

ing of tension and weight. There are *dyspnœa and cough*, described by Galen as the hepatic cough, slight, dry, and hard. The cough is supposed to be due to an impulse transmitted along the phrenic and vagus nerves. There may be *icterus* and *vomiting of bile.*

The symptoms continue to increase for eight or ten days. Fever persists and there is profuse perspiration. Death may result, or at this time the abscess forms and there is a *slight improvement in the general symptoms, with increase of the local symptoms* on the part of the liver.

In the *subacute form*, which is the most frequent in the temperate climate, the symptoms are less severe and the local symptoms of abscess come on more gradually.

The *chronic form* shows the greatest variations in symptomatology. Some cases show about the same symptoms as the acute and subacute forms, but not so severe, and the formation of the abscess is slower.

With the formation of abscess the *liver shows increase in size*, and a tumor may be visible. When the abscess is superficial the *thoracic walls may be œdematous.* The liver-dulness extends farther upward than normal. Often there is *pain radiating to the right shoulder.* There may be *jaundice* (frequently absent), sometimes *ascites* (even more rare than jaundice). There is *fever* as a rule, which varies in type and elevation.

Examination of the *urine* shows at first an increase of urea. Later there is hypoazoturia (Semmola and Gioffredi).

Diagnosis: Sometimes easy; sometimes exceedingly difficult. Malaria, typhoid fever, and tuberculosis must be ruled out. More difficult often is the differentiation from hyperæmia of the liver, echinococcus of the liver, and neoplasms. Some cases may be decided only upon puncture or incision.

The **prognosis** depends largely upon the form of the disease, better results being secured when there is a *single abscess* than when there are metastatic abscesses. The prognosis of metastatic abscess depends largely upon the primary disease. The prognosis should always be guarded. Spontaneous cure very seldom occurs.

Treatment is surgical. The medical treatment is symptomatic.

CIRRHOSIS OF THE LIVER (Chronic Interstitial Hepatitis; Hobnail Liver; Gin-drinkers' Liver; Contracted Liver).

Cirrhosis of the liver is characterized by general proliferation of the hepatic connective tissue.

Etiology: The most frequent cause is alcohol. Other toxic causes are lead, mussels (Segers), and spices and highly seasoned food (Budd), gout and diabetes, rickets and dyspepsia. Among the infections syphilis, tuberculosis, and malaria are causes. Some cases are probably due to *toxins*, especially the toxins of typhoid fever, measles, and scarlatina. Senility is a cause of cirrhosis of the liver associated with arterio-sclerosis and endarteritis. Some authorities believe that cirrhosis of the liver may result from retention of bile.

Adami found diplococci, sometimes resembling gonococci and sometimes appearing more like short bacilli, in the liver-cells and new connective tissue. Similar organisms were found by Adami in the Pictou cattle disease, an infective cirrhosis of cattle.

As a rule alcoholic cirrhosis is found in middle life. Barlow found cirrhosis of the liver, post-mortem, in a child eighteen months old who had received alcohol as food in the form of beer and gin.

Cirrhosis of liver—symptomatology: *The early symptoms are those of a gastro-intestinal catarrh:* eructations of gas, gastric pain, coated tongue, nausea and vomiting, and diarrhœa alternating with constipation.

There are *hepatic tenderness and pain radiating toward the right shoulder.* The patient is pale, emaciated, and experiences early fatigue. *At first the liver may be larger than normal,* but with the contraction of the cicatricial tissue the *liver becomes reduced in size* and the *surface uneven* (hobnailed).

Ascites comes on gradually and is frequently the symptom that causes the patient to seek medical advice. The ascites may be due to peritonitis (Hanot). Usually there is *enlargement of the spleen,* which often is difficult to detect.

The *subcutaneous abdominal veins become dilated*, especially on the right side, and there is dilatation of the capillaries along the margin of the ribs and around the umbilicus (*the caput Medusæ*).

The *urine* may at first be increased in quantity, but soon becomes scanty, high colored, and of high specific gravity. The reaction is strongly acid and urates are present in abundance. The amount of urea is decreased; urobilin and uric acid are increased. Semmola has observed a constant inverse ratio between the amount of urea and urobilin eliminated. There are peptonuria, sometimes glycosuria and albuminuria, from passive congestion and cachexia. With great diminution in the quantity of urine there may be symptoms of *toxæmia*. *Jaundice* more or less marked appears especially late in the course of the disease.

Hemorrhage is present in the majority of cases. Epistaxis is frequent. Hemorrhage from the gastro-intestinal mucous membrane, usually appearing as hæmatemesis, may be so great as to be fatal.

Fever may be present in acute cases, or may be caused by perihepatitis or catarrh of the bile-ducts.

Diagnosis: The history of long-continued indulgence in alcoholic beverages, with the presence of the symptoms of gastro-intestinal catarrh and ascites, would lead to the suspicion of *cirrhosis of the liver*. Confirmatory evidence would be furnished by physical examination. The liver is diminished in size, the surface nodulated; the spleen is increased in size, and there is cachexia. The surface-vessels are enlarged.

Cirrhosis of the liver should be *differentiated* especially from pyelophlebitis, pyelothrombosis, thrombosis, hypertrophic cirrhosis, diffuse chronic peritonitis, hyperæmia, amyloid liver, syphilis, carcinoma of the liver, and the simple atrophy of marasmus. Males are affected more frequently than females.

Prognosis is unfavorable as far as ultimate recovery is concerned. The usual duration of life is one or two years, although in some cases with judicious treatment life may be prolonged for a number of years. The course may be abruptly terminated by hemorrhage, pleurisy, bronchopneumonia, or tubercular peritonitis.

Cirrhosis of liver—treatment: The cause, if possible, should be removed. Alcohol, spices, highly seasoned food, and coffee must be withheld. The diet should consist of milk, broths, farinaceous foods in moderation, cooked fruits and vegetables, except potatoes. Carlsbad salt is useful for the relief of the gastro-intestinal catarrh.

In the treatment of ascites appeal is made to the diuretics. Digitalis may be given in combination with acetate of potassium. Hydragogue cathartics, compound jalap powder, gamboge, and elaterium may be used.

Sooner or later tapping (paracentesis abdominis) becomes necessary, and should be repeated as often as required. The withdrawal of the fluid must not be too rapid, or the distended peritoneal vessels may rupture and syncope result from cerebral anæmia.

Further medical treatment is purely symptomatic.

Hypertrophic cirrhosis of the liver: This variety of cirrhosis of the liver occurs especially between twenty-two and thirty-five, rarely after forty (Graham), most frequently in males. Differing from the ordinary cirrhosis of the liver, hypertrophic cirrhosis shows an *enlargement of the liver*, the *surface of which remains smooth* or is roughened only from a perihepatitis; *absence of ascites*, except in the later stages, when it is usually caused by peritonitis; *absence of enlargement of the subcutaneous abdominal veins*, except late in the course of the disease; a good appetite, only *slight emaciation*, and a somewhat lessened secretion of urea. The *duration* of the disease is usually longer than in the case of ordinary cirrhosis, frequently eight or ten years. The fæces contain bile, in the presence of icterus.

Treatment: The diet should be the same as in ordinary cirrhosis. Constipation must be overcome, best with the mild salines. Intestinal decomposition calls for calomel, hydronaphthol, and salol. Diuretics and the ingestion of milk increase the action of the kidneys and thus eliminate the poison that accumulates in the body through the disability of the liver.

Simple atrophy of the liver occurs in age and marasmus, is not a disease, and is not accompanied by characteristic symptoms.

ACUTE ATROPHY OF THE LIVER (Acute Yellow Atrophy; Icterus Gravis).

An **atrophy** of the liver characterized by destruction of the hepatic cells and severe jaundice.

Etiology: The disease is rare. Women are more frequently affected than men. The disease occurs frequently during pregnancy. It may occur at any age, from less than a year to over sixty, but is most common between twenty and thirty.

Symptomatology: The *early symptoms* are those of *gastro-intestinal catarrh:* loss of appetite, nausea, vomiting, constipation, pain and tenderness over the liver; later, *jaundice* in two-thirds of the cases, beginning in the face and gradually extending over the body. Sometimes there is an initial rigor. There may be general weakness, pains in the muscles, a tremulous tongue, and epistaxis. Later there are *cardiac asthma* and an *irregular pulse with increased tension.* Later marked *nervous symptoms* supervene—restlessness, delirium, coma, irregular breathing, which becomes stertorous, and death.

The *liver*, which at first may show some increase in size, becomes *greatly atrophied*, with diminished dulness from below upward and from left to right. Death may supervene before there is marked atrophy of the liver. Sometimes atrophy of the liver cannot be detected when there is an accompanying hyperplasia of the connective tissue.

Hemorrhages occur in more than half the cases, usually in the form of hæmatemesis.

Diagnosis: The early symptoms are those of catarrhal jaundice. The characteristic symptoms begin later: severe jaundice, hemorrhage, and nervous symptoms. Leucin and tyrosin are usually to be found in the urine. The objective symptoms, with the decreased hepatic dulness and increased size of the spleen, make the diagnosis clear.

The **prognosis** is unfavorable, although cases of recovery have been reported (Frerichs and Schnitzler).

The **treatment** is symptomatic. Cases of recovery have followed the use of aconite (Teissier) and benzoic acid and musk (Lebert).

Weil's disease: A severe relapsing febrile infectious icterus, believed by some to be a distinct disease. Others consider the disease only a form of febrile icterus. In some cases the typhoid bacillus has been found, which led to the belief that the disease is a "hepato-typhoid," but this view seems to have been disproved by finding the typhoid bacillus in cases which did not show the symptoms of Weil's disease (Dupré). In some cases a bacillus has been found, the proteus fluorescens, which Jaeger believes to be the specific infectious agent (see also under *Infections*).

FATTY LIVER.

Definition: The liver in the normal condition contains more or less fat. An abnormal deposit of fat in the liver is termed *fatty infiltration*, except when it is formed at the expense of the albumin of the organ, when it is known as *fatty degeneration*. Fatty infiltration and fatty degeneration may not always be readily differentiated, and often are associated.

Etiology: The amount of fat in the liver may be increased by eating fat and sugars, and by sedentary habits. Fat often accumulates in the liver in alcoholism and in many of the acute infections, very frequently in tuberculosis. Fatty degeneration takes place in acute yellow atrophy, phosphorus-poisoning, and has been produced experimentally in animals by the injection of the toxins of the bacillus pyocyaneus (Charrin), and by the injection of variola-poison (Roux and Yersin). There may be a localized fatty degeneration in cases of carcinoma.

Fatty liver—symptomatology: Light cases may show no symptoms. Pronounced cases show some enlargement of the liver, which does not present the normal solidity upon palpation. The surface of the liver is smooth and the border rounded. The increased weight of the liver may cause displacement downward. There is little or no bulging of the thorax.

Marked cases may show anorexia, vomiting, diarrhœa, and hemorrhoids.

The **diagnosis** of fatty liver is rendered probable by the presence of a uniform enlargement of the liver, with a smooth surface and rounded border, and with lessened resistance to pressure; and the absence of ascites, enlargement of the spleen, and jaundice. The history and physical examination may disclose the etiological factor.

Fatty liver should be *differentiated* especially from amyloid liver and leukæmia. Fatty liver frequently exists in combination with cirrhosis of the liver. The liver is then more solid upon percussion and the surface roughened, and there are ascites and enlargement of the spleen.

The **prognosis** depends largely upon the cause, and is greatly influenced by complications, especially fatty heart and fatty kidney. Except in the cases due to poisons or infections (acute yellow atrophy), life may not be cut short.

Fatty liver—treatment calls for removal of the cause, where this is possible. The treatment is largely symptomatic. Murchison recommends the internal use of large quantities of common salt.

AMYLOID LIVER.

Amyloid liver is usually associated with a similar involvement of the spleen, kidneys, and intestines. Amyloid matter is believed to be an albuminoid substance, which upon disintegration yields leucin and tyrosin.

Etiology: Amyloid liver has been observed at various ages, from two to seventy years, usually between twenty and thirty, more frequently in men than in women. The process is usually secondary to some chronic suppurative disease, especially tuberculosis and syphilis, and may follow malaria, leucocythæmia, pseudo-leukæmia, rickets, or gout.

Amyloid liver—symptomatology: As a rule the *liver is enlarged* and *firm upon pressure*, and the *surface is smooth*. The *bile* is diminished in quantity and poor in quality, with consequent intestinal disturbance and tympanites. There are always some *anæmia* and *leucocytosis*. There may be numerous symptoms

from affection of the spleen, kidneys, and intestines, and from the primary disease.

Diagnosis: Suspicion may be aroused by amyloid disease in other organs, especially the spleen, kidneys, or intestine. Characteristic of amyloid liver is the great enlargement of the liver, with firmness on pressure, rounded border, freedom from pain or tenderness upon pressure, except when pain is caused by perihepatitis or syphilis; and the presence of a chronic suppurative disease, tuberculosis, or syphilis.

The **prognosis** is unfavorable as a rule, but better where the liver alone is involved.

Treatment should address the cause, and is otherwise symptomatic.

NEOPLASMS OF THE LIVER.

Carcinoma of the liver: According to Eichhorst, carcinoma occurs, in the order of decreasing frequency, in the uterus, stomach, breast, and liver.

Occurrence: Carcinoma of the liver occurs most frequently at from forty to sixty years of age. The disease has been found in early life, even in the new-born child (Siebold). Carcinoma of the liver occurs more frequently in women, secondary to involvement of the uterus, ovaries, or breast. The disease is less frequent in hot than in cold countries. Usually carcinoma of the liver is secondary to carcinoma of the uterus or gastro-intestinal tract, especially the pylorus, cæcum, sigmoid flexure, or rectum; sometimes of the spine or right innominate bone. Carcinoma of the liver often follows traumatism from external violence or the irritation of biliary calculi.

The **symptoms** of carcinoma of the liver may be slight and indefinite, sometimes overshadowed by other disease. The *onset* is often insidious. *Emaciation* is marked, sometimes intense. Often there is *cachexia*. As a rule, the *liver is enlarged*, especially in young persons. The enlargement is sometimes sufficient to cause bulging of the thorax. The liver may feel *harder than normal*. Sometimes *nodules* may be detected by palpation. Often there is *pain radiating*

toward the right shoulder, through the connection between the phrenic and the fourth and fifth cervical nerves; and in the lumbar region. Among the *early symptoms* are anorexia, nausea, vomiting, and sometimes constipation. Diarrhœa may be present later. There may be *jaundice*, *tympanites*, and *ascites*.

Diagnosis: Differentiation between primary and secondary carcinoma of the liver may be difficult or impossible. The course of primary carcinoma of the liver is usually rapid and the enlargement of the liver more pronounced.

The *differential diagnosis* concerns hydatid cyst, sarcoma, abscess, and amyloid liver; syphilis of the liver; carcinoma of the pylorus, pancreas, mesentery, colon or kidney; and downward displacement of the liver.

The **prognosis** is unfavorable.

Treatment is palliative.

Adenoma of the liver resembles carcinoma in symptomatology, but the duration of life is longer. The *prognosis* is unfavorable. *Surgery* offers the only hope of cure.

Sarcoma of the liver occurs rarely as a primary disease, more frequently secondary to sarcoma elsewhere, especially in the region of the portal vein. As in sarcoma in other parts of the body, young persons are most frequently affected. The *symptoms* are similar to those of carcinoma of the liver.

Angiomata of the liver are small. In children they may attain some size. Usually they do not cause serious disturbance, and surgical treatment is unnecessary. Troublesome cases may demand resection of the liver.

Fibromata, *lipomata*, *gliomata*, and *cysts* occur occasionally in the liver.

DISEASES OF THE PANCREAS.

Hemorrhage into the pancreas may occur in acute pancreatitis or necrotic inflammation of the pancreas. Extensive hemorrhage may destroy the pancreas and invade the retroperitoneal tissue; or through a break in the peritoneal cover-

ing of the pancreas the hemorrhage may find its way into the lesser peritoneum.

The *symptoms* come on in the midst of apparent health, with severe pain in the upper part of the abdomen, with nausea and obstinate vomiting. Soon the patient becomes anxious, restless, and depressed. There are epigastric tenderness and sometimes marked tympanites. The temperature may be normal or subnormal. There may be constipation. These symptoms continue and the patient soon falls into collapse.

Treatment: Death is probably due to shock through the solar plexus (Zenker), and not to the loss of blood; and therefore it has been suggested that probably the best treatment would be to expose the pancreas and thereby relieve the pressure.

Pancreatic cysts are often due to traumatism or inflammation; but both these factors may be absent. Cyst of the pancreas has been observed in an infant six months old (Railton); but the great majority of cases occur between thirty and forty years of age.

The *symptoms* come on gradually, sometimes suddenly, as after traumatism, with attacks of colicky pain, nausea, and vomiting, and often with progressive enlargement of the abdomen. There may be glycosuria. Jaundice and dyspnœa may be caused by pressure. So-called pancreatic salivation, an increased secretion of saliva, is rare. Emaciation is sometimes marked. Transitory disappearance of the cyst has been reported.

Diagnosis: As a rule the cyst lies below the stomach and above the colon, and is affected little or not at all by respiration. The cystic fluid is alkaline in reaction; specific gravity, 1010–1020. Most important is the presence of ferments. The digestion of both fibrin and albumin is characteristic of the pancreatic secretion.

Treatment is surgical. Körte reports 101 cases in which the cyst was opened and drained, with a direct loss of only 4 cases. In 14 extirpations there were 12 recoveries.

Tumors of the pancreas: Carcinoma is the most frequent new growth. Much more rare are sarcoma, adenoma, and

lymphoma. The most important *symptoms* are epigastric pain, often paroxysmal in character; icterus, due to pressure; the presence of a tumor in the epigastrium, which may be difficult to detect; emaciation and cachexia, nausea and vomiting. Fatty diarrhœa and glycosuria are not common.

Treatment is surgical. Six recoveries in ten operations have been noted by Körte.

Pancreatic calculi, pancreatic lithiasis, is rare. The stones are white and usually numerous, as a rule composed of carbonate of lime, sometimes with phosphate of lime. Severe colic, glycosuria, and fatty diarrhœa were observed by Lichtheim in a case in which the diagnosis was confirmed by autopsy.

CHAPTER III.

DISEASES OF THE ORGANS OF RESPIRATION.

DISEASES OF THE NOSE.

Diseases of the exterior of the nose belong rather to the domain of dermatology or surgery. Those most commonly met are boils (furuncles); warts (verruca); acne; hypertrophy, which is sometimes wrongly termed lipoma, but resembles elephantiasis elsewhere; sebaceous tumors; nævus; rodent ulcer; lupus; rhinoscleroma; epithelioma; and injuries.

Of more interest to us are the diseases of the **interior of the nose.**

ACUTE CATARRH OF THE NOSE.

Acute rhinitis: An acute inflammation of the nasal mucous membrane, sometimes due to mechanical or chemical irritation, is usually *caused* by the action of bacteria or toxins. Iodine internally may cause iodism, manifested by coryza and the usual symptoms of a "cold."

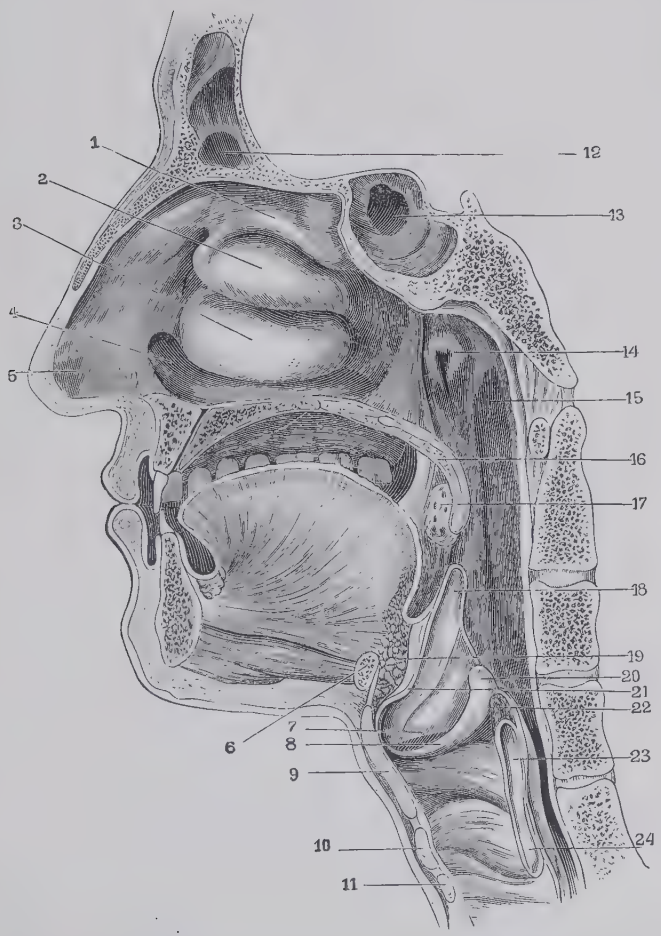
Attacks of acute nasal catarrh are frequently precipitated by changes of temperature, especially by the exposure of a portion of the body to cold and moisture. Bad ventilation is one of the most prominent causes.

Chronic inflammation of the nasal mucous membrane may predispose to acute attacks of nasal catarrh.

The **symptoms** of acute nasal catarrh, coryza, commonly known as a "cold," are too well known to need description. Aside from the local symptoms, there are general symptoms, usually ascribed to toxæmia.

The **diagnosis** of acute rhinitis is easy; but sometimes we may not readily locate the cause. The attack comes on

FIG. 29.



Vertical section of head, slightly diagrammatic. 1, superior turbinated bone; 2, middle turbinated bone; 3, lower turbinated bone; 4, floor of nasal cavity; 5, vestibule; 6, section of hyoid bone; 7, ventricular band; 8, vocal cord; 9, section of thyroid cartilage; 10, 23, and 24, section of cricoid cartilage; 11, section of first tracheal ring; 12, frontal sinus; 13, sphenoidal cells; 14, pharyngeal opening of Eustachian tube; 15, Rosenmüller's groove; 16, velum palati; 17, tonsil; 18, epiglottis; 19, adipose tissue behind tongue; 20, arytenoid cartilage; 21, tubercle of epiglottis; 22, section of arytenoid muscle (Seiler).

with malaise and chilliness; later there are some fever, loss of appetite, and pains in the joints. The swollen mucous membrane causes occlusion of the nasal passages. There may be frontal *headache* and affection of the eyes in severe cases. Soon there appears an acid discharge from the nose, usually with *sneezing*, sometimes with excoriation of the lip. With the beginning of the discharge the occlusion of the nose becomes less. Later the discharge changes to muco-purulent. The duration of the attack is about a week.

The **prognosis** is almost always good, but depends upon the cause.

Prophylaxis: As a rule "colds" are contagious. Feeble individuals should not be exposed to the danger of infection. Many cases would be prevented by attention to hygiene, especially personal cleanliness and proper ventilation. Coryza due to the administration of iodine (iodism) may be relieved by the discontinuance of the drug, or, where this is undesirable, by the administration of morphine.

Acute rhinitis—treatment: If possible, the cause should be discovered and removed. Defective hygiene must be corrected. The body should be kept clean.

The number of *remedies* is legion; space forbids even their enumeration. The nasal mucous membrane may be cleansed with an alkaline or astringent douche or spray, or with a cotton-wrapped sound. Increased nasal secretion may call for atropine. The toxic symptoms may be relieved by one of the coal-tar products, or opium in some form, best as Dover's powder. Often very great relief is afforded by the *hot bath*.

CHRONIC CATARRH OF THE NOSE.

Chronic rhinitis: Chronic nasal catarrh may be *caused* by an acute catarrh of the nose becoming subacute and later chronic. Thus the causes of acute catarrh of the nose, when long continued, may produce a chronic catarrh of the nose. Common causes are bad ventilation, dust, tobacco, and snuff.

The **symptoms** are less intense than in acute catarrh of the nose, and of longer duration. As in acute catarrh, the mucous membrane is swollen.

Diagnosis calls for differentiation from acute catarrh, polypus, and syphilis. Inspection reveals the mucous membrane swollen, especially over the turbinated bones, and covered more or less by secretion. There may be ulcers or erosions of the mucous membrane.

Prognosis: Sometimes chronic catarrh is quite obstinate to treatment, but persistence is usually rewarded by a cure.

Prophylaxis demands good hygienic surroundings and the avoidance of dust and the use of tobacco and snuff, things which play a prominent part in the causation of rhinitis.

Chronic rhinitis—treatment: The mucous membrane should be carefully cleansed. At first it is usually best to use an alkaline wash, and later an astringent solution.

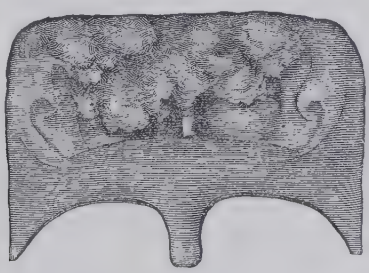
Bad cases may require a change of climate.

Syphilitic rhinitis: *Syphilitic catarrh* of the nose is characterized by lesions involving the deeper structures as well as the mucous membrane. Frequently there are evidences of syphilis elsewhere. Doubtful cases justify the therapeutic test.

NON-MALIGNANT NEW GROWTHS IN THE NOSE.

Polypi (myxomata) are the form of tumor occurring most frequently in the nose. The appearance of these growths

FIG. 30.



Adenoid hypertrophy at vault of pharynx (Lefferts).

has been likened to the pulp of a grape. At first they may cause sneezing and a thin, watery discharge. They cause more

or less occlusion of the nose and diminish or destroy the sense of smell. Numerous and diverse reflex disturbances are attributed to them. The voice is deadened. Frequently there are bronchitis and laryngitis.

The *diagnosis* is made by inspection, best after the application of cocaine. The *treatment* is surgical.

Other non-malignant tumors which may be found in the nose are fibromata, papillomata, angiomata, chondromata, osteomata, rarely cystomata. It is doubtful whether pure adenomata occur in the nose. *Adenoids* of the naso-pharynx (Fig. 30) may be mentioned. The *treatment* of all these tumors belongs to surgery.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

Acute inflammation of the laryngeal mucous membrane is usually due to *infection*, which is favored by some disturbance in the *nose*, obstruction, chronic inflammation; or *pharynx*, acute or chronic pharyngitis. Frequently mouth-breathing plays an important rôle in etiology. Other causes are the inhalation of impure air, dust, irritating fumes or vapors, excessive use of the voice, and certain exanthemata, especially measles and scarlet fever.

Symptoms: The voice becomes hoarse, there is dysphonia, sometimes aphonia. Cough may be present. General symptoms are absent or mild, unless there is at the same time involvement of other parts of the respiratory tract.

Diagnosis: Hoarseness, dysphonia, or aphonia should lead to a laryngoscopic examination, to find the cause of these symptoms. Such an examination would reveal a symmetrical inflammation of the mucous membrane of the larynx, bright red in color and swollen, the vocal cords pink; and would exclude other affections of the larynx, especially syphilis, tuberculosis, paralysis, and tumors.

Prognosis: As a rule, the disease lasts about a week; from five to eight days (Bosworth).

Prophylaxis calls for pure air, the proper treatment of dis-

eases of the nose and pharynx, and the avoidance of mouth-breathing. Singers and public speakers should avoid over-taxing the voice.

Acute laryngitis—treatment: Rest of the voice should be enjoined. Any nasal or pharyngeal disease should receive proper attention. It is better to confine the patient to a room that is comfortably warm, the air of which is kept moist with steam. The larynx may be cleansed with an alkaline solution and then treated with an astringent solution, such as a 1 per cent. solution of the liquor ferri persulphatis, or a 0.5 per cent. solution of nitrate of silver. These solutions are introduced upon a cotton-wrapped sound or in the form of a spray. Relief may be secured by the use of the steam atomizer. The application of cold to the neck, in the form of cold compresses, the ice-bag, or Leiter's coil, may be of value. The bowels should be kept open.

CHRONIC CATARRHAL LARYNGITIS.

Chronic catarrhal laryngitis may be due to a continuance of the causes of acute catarrhal laryngitis. Many cases depend upon deflection of the nasal septum or hypertrophic rhinitis. The disease is frequently found in individuals who use the voice excessively, and among those who indulge in alcoholic beverages.

Symptoms: The voice becomes husky and hoarse, especially upon exercise, singing, or speaking. There are numerous attempts at clearing the throat. As in acute catarrhal laryngitis, there may be cough. The absence of cough would indicate that the disease has not extended below the larynx. Rarely there is aphonia, which is usually temporary.

Diagnosis: The altered character of the voice, huskiness and hoarseness, should lead to a laryngoscopic examination, which would reveal a chronic inflammation of the larynx. The mucous membrane of the larynx is red and swollen, the blood-vessels injected. The vocal cords, instead of being glistening white, will appear grayish or pinkish, and will not approximate as well as in health.

The *differential diagnosis* is not always easy. Many cases

are due to tuberculosis and syphilis, and will be considered in connection with those diseases of which they form a part.

Prognosis: Spontaneous recovery does not occur, as in acute catarrhal laryngitis. Under treatment, which should include attention to any accompanying disease in the nose or pharynx, the affection may disappear and the voice return to its normal strength and clearness.

Prophylaxis is the same as for acute catarrhal laryngitis.

Chronic laryngitis—treatment: Diseases or malformations on the part of the pharynx or tonsils, which may have a causative relation to the laryngitis, should be removed. After cleansing the larynx with an alkaline wash, upon a cotton-wrapped sound or in the form of a spray, a 4 per cent. solution of ichthyol, a 1 per cent. or 2 per cent. solution of nitrate of silver, or some other astringent may be used. The value of resting the voice should not be overlooked. Erosions or chronic thickening of the mucous membrane may call for the local application of Lugol's solution and glycerin, 1 : 3, after the use of cocaine, 4 per cent., or a mixture of equal parts of cocaine, 4 per cent., and antipyrin, 10 per cent., which gives a more lasting anæsthesia. These applications may be repeated twice a week for a month or so. Obstinate cases may require curetting and the application of lactic acid under cocaine anæsthesia.

ŒDEMA OF THE LARYNX.

Etiology: The most frequent *causes* of œdema of the larynx are those which may cause dropsy elsewhere. Most important is disease of the kidney. Sometimes œdema of the larynx is caused by iodism, aneurism, or by a tumor pressing on the cervical veins.

Symptoms: The onset is usually sudden. The most striking symptom is inspiratory dyspnœa, which may become extreme in a few hours. Deglutition may be painful and difficult.

Diagnosis: The symptoms point to stenosis of the larynx. Upon laryngoscopic examination the laryngeal mucous membrane is found to be œdematous.

The **prognosis** depends upon the cause. In the absence of

treatment a case may terminate fatally in a few hours, from suffocation.

Treatment: Œdema of the larynx due to kidney-disease or cirrhosis of the liver may be relieved by free catharsis, which may be secured quickly by the administration of croton oil, gtt. j, or elaterium, gr. ss; and by free diaphoresis, which may be readily caused by the hypodermatic use of pilocarpine, gr. $\frac{1}{8}$, best given with alcohol internally to avoid depression.

A weak heart may need stimulation, best with the fluid extract of digitalis or tincture of strophanthus subcutaneously. The patient should be kept in a warm room, the air of which is kept moist with steam.

Tumefaction of the larynx may be relieved by scarification, which should be repeated if necessary. Severe cases may demand intubation or tracheotomy.

LARYNGEAL PERICHONDRITIS.

Laryngeal perichondritis is frequently found in connection with carcinoma, tuberculosis, syphilis, typhoid fever, diphtheria, pneumonia, erysipelas, and traumatism. Most cases are ascribed to exposure to cold or abuse of the voice.

Symptoms: After more or less malaise and chilly sensations, the attack comes on with headache, anorexia, sometimes with pain in the bones, and fever, 100°–101° F. Respiration, the use of the voice, and deglutition may be interfered with. Laryngoscopic examination reveals affection of the laryngeal cartilage.

Diagnosis: There are dyspnœa and the symptoms of acute inflammation. Laryngoscopic examination shows not only an acute inflammation of the larynx, but also an irregular swelling, usually upon one side. The cases due to tuberculosis run a chronic course; those due to syphilis are marked by pain and respond to the therapeutic test.

Laryngeal perichondritis should be *differentiated* especially from croup and acute submucous laryngitis. Croup shows an exudate. In submucous laryngitis the swelling is generally symmetrical and involves both sides.

Prognosis: The chief danger is through stenosis of the larynx, which may demand intubation. Cases due to tuberculosis or carcinoma have a bad prognosis. In all cases the course of the disease is long and tedious.

Laryngeal perichondritis—treatment: Cold may be applied to the neck and ice given internally. Sometimes relief is secured by scarification of the endolaryngeal tissues. Pain is relieved by the application of cocaine or the administration of morphine. The bowels should be kept open. Cases due to syphilis call for the use of the iodides. Bosworth advises the use of iodide of potassium during the acute stage, even in the absence of a history of syphilis. Dyspnœa may call for intubation or tracheotomy, or better laryngotomy. Sequestra should be removed, adhesions liberated, and strictures dilated.

SYPHILIS OF THE LARYNX.

Primary syphilitic lesion of the larynx occurs so rarely as to be considered a medical curiosity. The secondary lesions of syphilis may appear in the larynx as an erythema or a mucous patch, in four months to two years, usually within a year after the primary lesion.

More frequent in the larynx are the **tertiary manifestations** of syphilis: gummata, deep ulcerations, and cicatricial stenoses.

Prognosis: Under treatment the disease may be arrested; but destruction of tissue will be followed by cicatrization, which may lead to stenosis of the larynx.

The medical **treatment** is that of syphilis in general. Stenosis may demand dilatation, intubation, or tracheotomy.

TUBERCULOSIS OF THE LARYNX.

Laryngeal tuberculosis is usually secondary to pulmonary tuberculosis; but tuberculosis may be primary in the larynx. Here, as elsewhere, tuberculosis is due to infection by the tubercle bacillus.

Symptomatology: The voice becomes altered, weak, sometimes aphonic. The use of the voice requires great effort.

There may be an involuntary change from a low tone to a falsetto note, which may be maintained for a short time (Moure). The emaciation caused by the pulmonary tuberculosis, which usually precedes the affection of the larynx, is increased, and the expression of the patient becomes anxious. With extension of the disease, deglutition becomes difficult and painful. Destruction of the epiglottis may permit food to enter the larynx.

Diagnosis: A reaction to tuberculin or the presence of the tubercle bacillus in the sputum would be of little value in diagnosis, since in most cases there is tuberculosis of the lungs before involvement of the larynx. Cases of pulmonary tuberculosis may show alterations of the voice, due to non-tubercular affection of the larynx. Of most value in diagnosis is the laryngoscopic examination.

Tuberculosis of the larynx should be *differentiated* especially from syphilis and carcinoma.

The **prognosis** is grave. Eleven recoveries in fifteen cases have been reported by Heryng. The cases usually succumb to pulmonary tuberculosis. With improvement in the treatment of tuberculosis of the lung we may hope to save more cases of laryngeal tuberculosis.

Treatment: Probably of most value is the application of lactic acid or nitrate of silver. Orthoform may be used locally for the relief of pain. Climatotherapy and the use of tuberculin are important. Further than this the treatment is largely symptomatic (see Treatment of Tuberculosis). Operation may be justifiable, especially in the absence of pulmonary tuberculosis. Pain should be relieved by morphine and cocaine.

CARCINOMA OF THE LARYNX.

Carcinoma rarely affects the larynx. Men are affected more often than women. The disease occurs most frequently after fifty.

Symptomatology: The voice shows early impairment. There is dyspnœa. Cough is caused by the mucous or sero-mucous discharge. The breath is offensive (the odor has been described as musty), and there is more or less hemorrhage. As

a rule there are pain, sometimes difficult deglutition. Cachexia comes on late or may be absent.

Early **diagnosis** is difficult or impossible. Later the symptoms, the peculiar laryngoscopic appearance, and the progressive course of the disease may render the diagnosis more or less absolute. In doubtful cases a positive diagnosis may be made by a microscopic examination of a portion of the growth.

Prognosis: The disease is almost absolutely fatal. Cases of apparent cure by operation have been reported (Billroth, Butlin). Bosworth gives the fatality of operation at over 90 per cent.

Treatment: Early and complete removal of the growth is important. It must be remembered that the process extends beyond the apparent infiltration of the lymphatics. A great obstacle to operation is offered by the difficulty experienced in making an early diagnosis. Otherwise the treatment is symptomatic.

SARCOMA OF THE LARNYX.

Sarcoma occurs in the larynx very rarely. The majority of the reported cases have occurred in men. The ages of the patients have ranged from nineteen to seventy-four years, most of the cases occurring between forty and sixty.

Symptomatology: The voice becomes hoarse, sometimes aphonic. There are dyspnœa, cough, sometimes dysphagia. There may be slight hemorrhage and some pain. Late in the course of the disease there may be some cachexia.

Diagnosis: Suspicion of malignancy may be aroused by the symptoms and laryngoscopic examination. A positive diagnosis may be made by microscopic examination.

A point in the *differential diagnosis* from carcinoma is the involvement of the cervical glands late in carcinoma, which is usually absent in sarcoma.

The **prognosis** is grave. Very few recoveries have been reported.

Treatment: The only hope of cure lies in operation. All other treatment is palliative.

BENIGN TUMORS OF THE LARYNX.

Benign tumors are found in the larynx much more frequently than malignant growths. Bosworth gives the order of frequency as follows: papillomata, fibromata, cystomata, myxomata, adenomata, lipomata, angiomata, enchondromata, and mixed tumors. It is doubtful whether pure adenomata occur in the larynx.

The chief **symptoms** are interference with phonation and respiration.

The **diagnosis** is made with the laryngoscope. *Differential diagnosis* may call for the use of the microscope.

The **prognosis** is usually good. These tumors rarely offer serious interference to respiration.

Treatment, where necessary, is surgical.

Neuroses of the larnyx: The chief neuroses of the larynx are: paralysis, spasm of the glottis (laryngismus stridulus), muscular incoördination, neuralgia, hyperæsthesia, paræsthesia, anæsthesia, and hysterical aphonia.

DISEASES OF THE TRACHEA AND BRONCHI.

The **trachea** and **bronchi** are rarely affected primarily. Diseases of the trachea and bronchi usually come from above, the larynx; or from below, the bronchial tubes; and in most instances the disease of the trachea or bronchi is overshadowed by the primary affection. Malignant disease of the trachea and bronchi is usually secondary.

Summary: The mucous membrane of the trachea may be inflamed to constitute an *acute* or *chronic catarrhal tracheitis*. *Diphtheria* has been reported to occur primarily in the trachea; but in the great majority of cases it is secondary to invasion of the larynx. The trachea or bronchi may show *ulceration*, acute or chronic, and *stenosis*. *Tumors* of various kinds may occur in the trachea or bronchi; or these organs may suffer *compression*, due usually to diseases in the thyroid gland, the mediastinal glands, the vertebræ (tuberculosis), œsophagus, aorta (rarely due to large pericardial effusion).

The *diagnosis* of diseases of the trachea and bronchi may be difficult. In such cases the use of the laryngoscope may give valuable information, but the examination is much more difficult than inspection of the larynx.

BRONCHITIS.

Etiology: Bronchitis is due to infection in the vast majority of cases, either directly through the invasion of the bronchial mucous membrane by micro-organisms; or indirectly, through the elimination of toxins, as may be observed especially in typhoid fever and cerebro-spinal meningitis. Bronchitis may also be caused by the elimination of poisons other than the toxins referred to, especially iodine and alcohol. Many of the exanthemata, particularly measles and smallpox, show bronchitis. Direct invasion of the bronchial mucous membrane usually is an extension of an inflammation from the upper respiratory passages, the mouth or nose, or may be caused by trauma.

ACUTE BRONCHITIS.

Definition: An acute inflammation of the bronchial mucous membrane.

Etiology: Most cases are attributed to "catching cold." "Catching cold" usually occurs in badly ventilated apartments, rather than in the open air. Sometimes the disease is due to mechanical or chemical causes, dust, or irritating fumes. An acute bronchitis is often found in connection with the infections, especially measles and the respiratory form of influenza. The most prominent predisposing causes are tuberculosis, syphilis, rheumatism, gout, diabetes, Bright's disease, cancer, and heart-disease. An acute bronchitis appears in some individuals following the use of even small quantities of iodide of potassium. Acute bronchitis occurs most frequently in the colder months, and especially at the extremes of life. The disease is very common among the users of alcoholic beverages, probably due to elimination through the bronchial mucous membrane. Acute bronchitis is often caused by too little exercise in the open air.

Acute bronchitis—symptomatology: Usually there are the *general symptoms of an infection*—chilly sensations, fever, increased pulse-rate, malaise, anorexia, headache, often a coated tongue, more or less constipation, and in severe cases there may be pain in the limbs. The last-mentioned symptom would seem to indicate a *toxæmia.*

The *early local symptoms* are *dryness and constriction* in the region of the larger bronchial tubes, the bronchi and trachea, frequently with *hoarseness* and a dry *cough*, from involvement of the larynx and trachea. There may be dyspnœa. In a few days the exudation from the bronchial mucous membrane becomes more profuse. There is *expectoration.* Light cases may last but a week or two.

In *more severe cases* there may be sleeplessness and prostration. In the aged the temperature may be subnormal. As a rule the dyspnœa becomes greater the further the inflammation extends toward the air-cells, amounting sometimes to orthopnœa. Children may have convulsions.

The chief *complications* are inflammation of the upper air-passages, laryngitis and tracheitis, and atelectasis and broncho-pneumonia.

Diagnosis and physical signs: Percussion may be negative, or may reveal dulness in the presence of atelectasis. Expiration is prolonged. Subcrepitant râles may be found when the inflammation involves the smaller ramifications of the bronchial tubes. These may be heard on both sides, especially at the base of the lungs.

Differential diagnosis calls especially for the recognition of pneumonia or broncho-pneumonia.

Of most importance as a rule is the recognition of the cause of the bronchitis, especially tuberculosis, syphilis, rheumatism, gout, diabetes, Bright's disease, measles, and whooping-cough.

Prognosis: The mortality increases as we approach the extremes of life, and also as the inflammation advances along the finer bronchial tubes toward the air-cells. More deaths occur in winter than in summer. Mild cases usually recover in a week or two A fatal result does not often occur among robust adults and children. The prognosis assumes gravity

with the cause, the condition of the patient, and the severity of the attack.

Acute bronchitis—treatment: The patient should occupy a warm, well-ventilated room, exposed to the sun. The air of the room may be kept moist with steam, or the patient may obtain relief by the use of a *steam atomizer*, or by inhaling steam from a kettle or pitcher. The compound tincture of benzoin may be added to the water. *Catharsis* should be secured by a mercurial or saline laxative. *Diaphoresis* is sometimes of value. There are a large number of *expectorants*. As a rule the best are apomorphine, ipecac, and squills. Cough and insomnia may be relieved by the use of codeine, morphine, or opium, best in the form of Dover's powder, which may be given in a syrup. *Hot applications* may be made to the chest, or turpentine or a weak mustard plaster may be used. Rubbing the chest with a liniment may secure some relief, at least of the mind of the patient. An *emetic* is sometimes useful in the case of infants who may not be able to expectorate. The aged may require alcohol, senega, and carbonate or chloride of ammonium. *Inhalations of oxygen* are recommended, especially at the extremes of life. During convalescence, tonics, fresh air, and exercise are of value.

Where the bronchitis is secondary to syphilis, rheumatism, or diseases of the lungs, heart, kidneys, etc., the treatment must address the primary disease.

Capillary bronchitis: The term has been applied to an acute bronchitis affecting the finer bronchial tubes. Such a division of bronchitis is an over-refinement. It is difficult to imagine a case in which such an inflammation would not extend to the air-cells to constitute a broncho-pneumonia.

CHRONIC BRONCHITIS.

Definition: A chronic inflammation of the bronchial mucous membrane.

Etiology: Chronic bronchitis may result from an acute bronchitis, especially when the attacks of acute inflammation are frequently repeated. Chronic bronchitis is also found in

tuberculosis, emphysema, asthma, disease of the heart, especially stenosis or insufficiency of the mitral valve; rheumatism, gout, diabetes, alcoholism, or where almost any of the causes of acute bronchitis, such as the inhalation of dust, are long continued. To some of the causes of acute bronchitis tolerance may be established before the production of chronic bronchitis. Chronic bronchitis is usually found in middle or advanced life.

Chronic bronchitis—symptomatology: The onset is gradual. The symptoms improve in summer, to become aggravated in winter. As in acute bronchitis, there may be dyspnœa and discomfort under the sternum. There is more or less cough, which in bad cases may become violent. The cough may cause insomnia. Sputum may be almost absent, or present in varying amounts, sometimes constituting a bronchorrhœa. There may be fetor, usually due to sputum retained in dilated bronchi. Sometimes the bronchi are not dilated.

The *physical signs* resemble those of acute bronchitis. The duration of the disease is indefinite, but longer than in acute bronchitis.

The principal *complications* are atelectasis, broncho-pneumonia, emphysema, bronchiectasis, and dilatation of the heart, usually of the right side of the heart.

Diagnosis: The history and symptoms render valuable aid. Chronic bronchitis should be differentiated, especially from pneumonia and tuberculosis. Chronic bronchitis differs from pneumonia in being a bilateral affection without evidence of consolidation and with little or no fever. Tuberculosis usually shows a more marked decrease of weight and greater weakness, and, as a rule, the tubercle bacillus may be found in the sputum. In doubtful cases the differential diagnosis may call for a test-injection of tuberculin.

Prognosis: Much depends upon the cause of the bronchitis, the severity of the disease, and the strength of the patient. Chronic bronchitis is most dangerous in the feeble and aged. The prognosis should be guarded in the presence of emphysema, bronchiectasis, or dilatation of the heart.

Chronic bronchitis—treatment: The general or curative treatment of bronchitis must address the underlying cause, what-

ever that may be. All other treatment is palliative or symptomatic.

Bronchitis due to cardiac insufficiency may be relieved by purgation, diaphoresis, and the use of stimulants, digitalis, strophanthus, alcohol, and nitroglycerin.

Bronchitis due to pressure from an aneurism or tumor may be relieved by opium. Iodide of potassium may be of value.

Rheumatism or gout should be properly treated with the salicylates, Carlsbad salts, colchicum, etc. (see Rheumatism and Gout). Where bronchitis is due to the inhalation of dust, irritating vapors or fumes, a change of occupation may be necessary. Tuberculosis, pleurisy, disease of the liver, or any other disease upon which the bronchitis may depend should receive proper attention.

For the dry catarrh, the "catarrhe sec" of Laennec, opium, best in the form of codeine, morphine, paregoric, or Dover's powder, often affords great relief, but should not be used when there is high fever or great prostration. Chronic bronchitis is a disease of long duration, and opium may not be indefinitely continued. Heroin, or heroin hydrochloride, in doses of 0.005–0.015 gm., diminishes the desire to cough, deepens and prolongs respiration, and relieves pain. Chlorate of potassium is a good expectorant. Various other sedatives, narcotics, and expectorants are recommended. Much relief may be obtained from the inhalation of steam.

Where the secretion is excessive and the cough unavailing, stimulating expectorants may be useful, such as senega, which may be given in teaspoonful doses of the simple syrup of senega, or gtt. xxx of the compound syrup of squills; carbonate and chloride of ammonium, balsam of copaiba, and the various preparations of turpentine; syrup. picis liq., ʒij-iv t. i. d.; myrtol, turpentine, terebene, and terpene hydrate, ℳv t. i. d. Cubebs may be given in cough-lozenges. Apomorphine is an excellent expectorant, but should not be given when there is a weak heart. Iodide of potassium may be given, gr. v-xxx ter die. Often very great comfort is secured from the use of the steam atomizer, in which various sub-

stances may be used.[1] Sometimes a change of climate is advisable. Cases of dry bronchitis are usually benefited most by a warm, moist climate, such as may be secured in the Bermudas, Nassau, Florida, Southern California, the Azores, or Madeira.

FIBRINOUS BRONCHITIS (Plastic Bronchitis).

Definition: An inflammation, acute or chronic, of the bronchial mucous membrane, characterized by the formation of a fibrinous exudate in the bronchial tubes. The disease has been found in the new-born child on autopsy (Hayn). Fibrinous bronchitis, is most frequent between ten and forty years. The condition is rare in the aged.

The **etiology** of the disease is obscure. Escherich (1883) failed to find the bacillus of diphtheria in the exudate. Three varieties of micrococci were isolated by Picchini (1889). Many cases have been observed to follow traumatism or chemical irritation. Exposure to cold and moisture is frequently given as a cause. Among the predisposing or underlying causes the following have been observed: tuberculosis, syphilis, alcoholism, rickets, pregnancy, and typhoid fever.

Fibrinous bronchitis—symptoms: There are present the symptoms of an acute or chronic bronchitis. At times cough and dyspnœa become intense, to be relieved by the expulsion of a fibrinous bronchial cast. These casts are branched like a tree, corresponding to the ramifications or branches of the bronchial tubes from which they are expelled and of which they form a cast. The casts vary in size, usually an inch to an inch and a half in length, rarely reaching a length of four inches or over. Hæmoptysis is a common symptom.

[1] Mason, in the *American System of Practical Medicine*, gives the following list of substances that may be used with the atomizer, with the quantity of each to be added to one ounce of water:

Tincturæ iodii, ♏ij–x;
Acidi carbolici, gr. ij;
Creosoti, ♏iij;
Acidi tannici, gr. ij–x;
Alumini exsiccati, gr. iij–xv;
Liquoris ferri subsulphatis, gtt. v–xx;
Tincturæ opii, ♏v–xxx;
Tincturæ opii camphoratæ, ʒj–iij;
Morphinæ sulphatis, gr. ss–j;
Solut. cocainæ hydrochlorici (4 per cent.), ♏xxx–lx;
Tincturæ hyoscyami, ♏xxx–lx;
Tincturæ stramonii, ♏xxx–xl;
Tincturæ belladonnæ, ♏xxx–xl.

Diagnosis: Finding the peculiar casts makes the diagnosis. A localized subcrepitant râle may be suggestive (Flint). Blood-casts may appear in cases of hæmoptysis, and should not be mistaken for the casts characteristic of fibrinous bronchitis. Acute pneumonia and diphtheria also may show casts.

Prognosis: Fibrinous bronchitis is most dangerous at the extremes of life. Death is most frequently caused by complications, or the underlying diseases which predispose to fibrinous bronchitis. Extension of the exudate into the trachea or inability to expel casts may cause death by suffocation. Aside from other diseases of the lungs, especially tuberculosis, the prognosis is usually good.

Treatment: Of most value are inhalations of steam and the use of expectorants, particularly after the cast becomes loose. Probably the best expectorant in these cases is apomorphine. Iodide of potassium seems to be of little or no value.

BRONCHIECTASIS (Dilatation of the Bronchial Tubes).

Etiology: Most of the cases are probably due to weakening of the walls of the bronchial tubes, caused by chronic bronchitis. Many cases are caused by whooping-cough, measles, tuberculosis, asthma, and pleurisy. Sometimes cases may be caused by obstruction of the air-passages by foreign bodies, enlarged glands, tumors, aneurisms. Rarely the condition is congenital.

The **symptoms** in cases of slight or moderate dilatation may not be characteristic. Marked dilatation of the bronchial tubes may be followed at times, especially in the morning, by expectoration of large quantities of muco-purulent sputum, often fœtid in character.

On **physical examination** percussion may reveal the presence of cavities. The signs of bronchitis are usually present.

Diagnosis: In slight or moderate cases of bronchiectasis diagnosis may be impossible during life. In doubtful cases the diagnosis may call for an exploratory puncture. Bronchiectasis should be *differentiated* especially from tuberculosis, actinomycosis of the lung, pulmonary gangrene or abscess, and empyema. The diagnosis of tuberculosis or actinomycosis of

the lung may be established by an examination of the sputum for the tubercle bacillus and ray fungus respectively. Gangrene and abscess of the lung show more pronounced general symptoms than are present in bronchiectasis. Bronchiectasis, barring complications, shows little or no fever and only slight general symptoms, except in cases that are far advanced. An empyema that discharges through the lung may closely simulate bronchiectasis, but usually shows fever, and sometimes pneumococci may be found in the sputum.

The **prognosis** is best in childhood; worst in the weak and aged, especially in the presence of consolidation or collapse (atelectasis) of the lung-tissue.

Bronchiectasis—treatment: Some cases improve under the use of iodide of potassium, probably only when syphilis plays a rôle in etiology. Some relief may be afforded by inhalation or administration of turpentine, creosote, tar, menthol, eucalyptus, myrtol. Some cases have been successfully treated surgically by incision and drainage; but surgery does not offer as much hope as in the treatment of abscess of the lung. In most cases of bronchiectasis that have been operated upon the operation has only hastened a fatal termination.

ASTHMA.

Definition: A peculiar *dyspnœa*, characterized by difficult and prolonged expiration, hyperæmia of the bronchial mucous membrane, more or less acute emphysema of the lung, and sibilant râles. The sputum often shows Charcot-Leyden crystals and Curschmann spirals.

Etiology: Depending upon the cause, asthma is divided into (1) *primary asthma*, sometimes called bronchial asthma or pulmonary asthma; and (2) *secondary asthma*, which is subdivided into cardiac asthma, renal asthma, etc. With the advance of our knowledge of the etiology of asthma the number of cases of primary asthma are diminishing, while the secondary asthmas are increasing.

Asthma is supposed by some observers to be due to *contracture of the bronchial muscles*, through some affection of the nervous system, the cause of which is known in secondary

asthma and unknown in primary asthma. Asthma is more frequent in men than in women.

Cases which have seemed to depend upon *swollen* tracheal or bronchial *glands* have been explained by the supposition that such enlargements cause irritation of the vagus nerve through pressure.

An important rôle has been ascribed to a *special susceptibility* of the *nervous system* that in some individuals causes an asthmatic attack to follow stimuli that in other individuals would be without such effect. In many cases *heredity* seems to play a part. Asthma may alternate with other neuroses, such as epilepsy, hemicrania, angina pectoris.

Nasal polypi and other affections which interfere with the respiratory function of the nose are a frequent cause of asthma. Such cases have been ascribed to *reflex irritation.*

In some instances the attacks are observed to occur only during the *menstrual period.*

Cullen gives the account of an apothecary's wife who had an attack when ipecac was handled in the shop. Trousseau had an attack in the presence of a bouquet of violets. Itzigson records the case of a merchant who would have an attack when fresh coffee was handled in his presence. Mackenzie reports the case of a lady who had an attack upon seeing a rose, even though it were artificial (*psychic asthma*).

Most cases of asthma occur at night, often regularly at the same hour. In some cases the attacks will not appear if a light is left burning.

Many cases of asthma seem to bear a relation to *gout*, and in some cases a seeming relation with *chronic skin diseases* (herpes, psoriasis, and eczema) has been reported.

Symptomatology: Asthmatic *attacks occur suddenly* at irregular, sometimes regular, intervals, usually in the *night*-time, with *intervals of apparent perfect health.* The *attack* is characterized by severe *dyspnœa*, calling for the use of all the accessory muscles of respiration. The difficulty is with *expiration*, which is prolonged. The hunger for air causes the patient to assume a posture that will give freedom and power to the accessory muscles of respiration. There is *cyanosis.* The attack may continue for a few minutes to a few

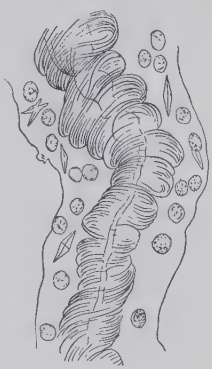
hours, when the symptoms gradually, sometimes suddenly, disappear.

Laryngoscopic examination shows the mucous membrane of the trachea and visible bronchi reddened.

Physical signs: Percussion reveals an *increased lung-area*, the border of the lungs extending further downward, with a lower position of the liver. The heart-dulness is diminished. Upon *auscultation*, *sibilant râles* are heard, replaced toward the end of the attack by *moist râles*. A *vesicular respiratory murmur* is heard over parts of the lungs.

Fever is absent, or, if present, would denote complication. In children especially a rise of temperature is often due to catarrh. *Expectoration* usually occurs only toward the end of the attack. The frothy, grayish-white *sputum* contains the *Charcot-Leyden crystals* and *Curschmann spirals* (Fig. 31). Spirals have been found also in pneumonia, fibrinous bronchitis, acute and chronic catarrhal capillary bronchitis, diseases which affect the smaller bronchi and bronchioles. The sputum contains *eosinophile and granular cells* (Mastzellen), and crystalline and amorphous phosphate of lime. During the attack large numbers of eosinophile cells have been observed in the blood by many observers. Other observers have failed to confirm this finding.

FIG. 31.



Spirals and crystals in sputum of asthma.

The more common **complications** are bronchitis, gout, diseases of the skin (herpes, psoriasis, eczema), epilepsy, neuralgia, pulmonary emphysema, and bronchiectasis. Tuberculosis may occur in an asthmatic patient, but is not common.

Diagnosis: Asthma is characterized by paroxysmal expiratory dyspnœa. The sputum usually contains Curschmann spirals, Charcot-Leyden crystals, and eosinophile cells. During the attack there are an acute emphysema and sibilant, later moist, râles. Not all dyspnœas are asthmas.

Prognosis: Where the cause can be discovered and removed

the case may be cured. Frequently treatment is followed only by a cessation of symptoms, which may last even for years, and finally return. Cures are more frequent in early life. Asthma rarely causes death.

Asthma—treatment: *Treatment of the attack:* Any discoverable cause should be removed. Attacks may be cut short by the use of opium, morphine, or chloral; but these remedies may not be used continuously. Belladonna, atropine, cannabis Indica, and strychnine may be used. Chloroform, ether, methylene bichloride, and ethyl iodide may be inhaled, but have only a transitory effect. The leaves of stramonium and belladonna have long been in use, smoked either with or without tobacco. A good combination is the following, given by Trousseau as the composition of the "cigarettes Espic:"

℞	Fol. elect. herb. belladonnæ,	0.36;
	Fol. elect. herb. hyoscyami,	0.18;
	Fol. elect. herb. stramonii,	0.18;
	Fol. elect. phellandrii aquat.,	0.06;
	Extract opii,	0.008;
	Aquæ laurocerasi,	q. s.

This is made into a cigarette and one or two such cigarettes may be smoked during an attack.

In some cases those who are not accustomed to the use of tobacco may gain much benefit from its use. Arsenic and nitre are also used, blotting-paper being soaked in a solution of these substances, then dried, and smoked or burned and the fumes inhaled. The inhalation of ammonia is often of value. Electricity is sometimes used, the induced or faradic current.

Cases due to nasal irritation may be relieved by the local application of cocaine.

Treatment during the intervals: Often a local cause in the upper respiratory organs or in the genital organs may be disclosed and treated or removed, when the symptoms will disappear. Sometimes several points of irritation may be found. The iodides may be given internally, 1.5–3.0 per day, prescribed in peppermint-water and taken largely diluted in milk. The remedy must be given for a long time. Fowler's solution of arsenic is often of value. Numerous remedies

and contrivances have been recommended. In each case the physician should seek and treat or remove the cause. This sometimes may call for a change of residence. Good hygienic surroundings and exercise, especially open-air respiratory gymnastics and hydrotherapy, are often of very great value.

DISEASES OF THE LUNGS.

PNEUMONIA.

Definition: An infection of the lung by various micro-organisms, the invasion of which may be favored by exposure to inclement weather, trauma, etc.

Varieties: (1) croupous pneumonia, lobar pneumonia, fibrinous pneumonia, sometimes referred to as genuine pneumonia; (2) catarrhal pneumonia, lobular pneumonia, broncho-pneumonia. To these may be added (3) influenza pneumonia, due to the influenza bacillus; (4) tubercular pneumonia, due to the tubercle bacillus, and really a tuberculosis; (5) true typhoid pneumonia, due to invasion of the lung by the typhoid bacillus. The term typhoid pneumonia has been abused so much that many suggest that the term should be dropped altogether. (6) Septic pneumonia, set aside by some observers as a special variety, due to the pus-producing micro-organisms. Such cases, when possible, may be classified according to the particular variety of micro-organism present, as streptococcus pneumonia, staphylococcus pneumonia, etc.

CROUPOUS PNEUMONIA (Lobar Pneumonia; Fibrinous Pneumonia; Genuine Pneumonia).

Definition: An infection of the lung, affecting an entire lobe, characterized by a fibrinous exudate with rusty-colored sputum, high fever, and termination by crisis in five to nine days.

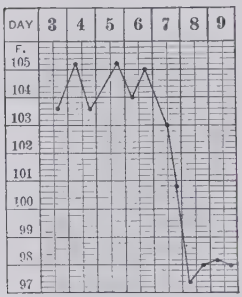
Etiology: The infectious agent is the *micrococcus pneumoniæ crouposæ* (Sternberg) or the bacillus of Friedländer. These micro-organisms have been found upon the respiratory mucous membrane, especially in the mouth and throat, in

health, and it would seem that exposure to cold and moisture and trauma may play an important *rôle* in etiology. In some cases other micro-organisms have been found, such as the influenza bacillus, streptococcus pyogenes, staphylococcus pyogenes aureus, and the typhoid bacillus; but in such cases the disease does not pursue the typical course of croupous pneumonia.

Croupous pneumonia—symptomatology: Some cases show prodromata for two or three days: malaise, more or less inflammation of the respiratory mucous membrane, especially of the nose and pharynx, and indigestion.

Usually there are no prodromata. The disease is announced suddenly with a *chill, followed by fever*, the temperature reaching 104° or 105° F. As a rule, sooner or later there is *pain in the side*, usually in the region of the nipple, caused by involvement of the pleura. *Dyspnœa* is prominent, due to pain or to the congestion of the lung.

Fig. 32.



Temperature-curve of croupous pneumonia.

All sorts of *râles* may be heard, coarse and fine, moist and dry. This constitutes the stage of engorgement, which in a day or two gives way to consolidation, often with relief of the pain and dyspnœa.

The temperature continues high, with rapid pulse and respiration, anorexia, thirst, headache, constipation. The *urine* is reduced in quantity and highly colored. The *sputum becomes rusty-colored.* Usually the cough is painful. There are restlessness and more or less delirium.

As a rule, between the fifth and ninth day *resolution* is announced by a sudden fall of temperature, *crisis*, with profuse perspiration (Fig. 32). Occasionally the temperature falls by lysis, reaching the normal in a few days instead of a few hours. The *pulse* falls from 110 or 120, sometimes 150 in children, to 50 beats per minute.

Croupous pneumonia—physical examination: *Inspection* re-

veals lessened expansion of the affected side. *Palpation* may detect *increased vocal fremitus* and sometimes a pleuritic *friction-sound*. *Percussion* usually shows increased *resonance* over the affected lobe during the period of congestion, which during the period of hepatization (consolidation) gives way to *dulness*. After resolution resonance reappears. *Auscultation* discloses both fine crepitant and coarse *râles* during the period of congestion. The former disappear during consolidation of the exudate. The *breathing then becomes bronchial*. There is *bronchophony*, sometimes ægophony, over the affected lobe. With resolution, bronchial breathing and bronchophony give way to the *crepitus redux*, moist râles which are usually coarser than the fine moist râles heard during the period of congestion. In *central pneumonia*, in which the affection of the lobe does not extend to near the periphery, auscultation may reveal only bronchophony.

Especially in severe cases, the heart is called upon to do increased work and the cardiac dulness is found to extend further to the right. Usually there is accentuation of the second pulmonary valve sound. There is often enlargement of the spleen and liver.

Croupous pneumonia—examination of the blood: There is a marked *leucocytosis*, 20,000–32,000 (Ewing). In a very virulent case as high as 100,000 has been recorded (Kidd). As a rule, a low number, below 14,000 (Ewing), lends gravity to the prognosis. A very high number is found in severe cases. The number of leucocytes gradually diminishes just before crisis and returns to the normal after resolution. An increase in the leucocytosis would indicate a further invasion of the pulmonary tissue.

Complications: Bronchitis and pleurisy occur frequently with croupous pneumonia. Sometimes there is empyema. Pericarditis is found most frequently in pneumonia of the left lung. Occasionally there are endocarditis, meningitis, nephritis; more rarely peripheral neuritis, urethritis, parotitis, and orchitis.

Diagnosis: Usually the symptoms and physical examination render the diagnosis easy. The disease comes on suddenly, with increased respiration, sometimes localized pain in the

region of the nipple, with cough, later rusty sputum and physical evidences of consolidation of the lung upon the affected side.

Pneumonia of the *apex* of the lung may resemble tuberculosis, but does not show the tubercle bacillus in the sputum nor respond to the test with tuberculin. In such cases, as well as in cases of central pneumonia, in which the physical signs may be absent or misleading, an examination of the blood will show leucocytosis. *Acute pulmonary œdema* may show dyspnœa, cyanosis, râles, and sometimes sputum somewhat resembling that of pneumonia. Acute œdema usually depends upon disease of the heart, and is not accompanied by high fever. *Pleurisy* usually comes on more gradually and does not show rusty sputum. Doubtful cases may call for aspiration. Pluerisy and croupous pneumonia may co-exist.

Prognosis: Much depends upon the condition of the heart. The prognosis should be extremely guarded when the heart is enfeebled by age, alcoholism, or disease. The occurrence of complications adds gravity to the case. Pneumonia occurring in pregnancy, especially in the later months, frequently causes miscarriage and a fatal termination. In any case marked and persistent increased frequency of the pulse and respiration, the expectoration of "prune-juice" sputum, persistent tracheal râles, the typhoid state with low delirium, stertor, muscular tremor, and coma, are ominous signs.

Croupous pneumonia—treatment: In the way of specific medication most promising are the results that have been obtained by the injection of blood-serum from recent convalescents. Frequently crisis occurs immediately or soon after such injections. The effectiveness of serum-therapy is ascribed to an antipneumotoxin, which normally accumulates in the body of the patient to cause the crisis on the seventh to the ninth day of the disease. We should not forget, in the application of serum-therapy, that all cases of pneumonia are not due to the same micro-organism.

In general the treatment is symptomatic. The sick-room should be well ventilated. Some temperature belongs to the disease and is salutary; temperature above 103° F. calls for hydrotherapy, best sponging with cold water, and the use of

ice upon the chest. Severe pain may be relieved by morphine; cough that is distressing, by Dover's powder. Nervous symptoms – headache, sleeplessness, delirium—may call for sponging with cold water or the application of the ice-bag or cold compresses to the head, or the administration of Dover's powder or trional at bedtime.

Most important is the *support of the heart.* A flagging heart calls for the use of alcohol and strychnine. Nitroglycerin or musk may be used to bridge over a threatened collapse. Digitalis or strophanthus may be indicated by weakness of the heart. Some brilliant results have been reported from the use of large doses of digitalis or digitalin; but others have failed to secure such results by the use of these remedies in pneumonia.

Good results have been reported (Lepine) from the intrapulmonary injection of bichloride of mercury, 20–26 c.c. of a 1 : 4000 solution.

CATARRHAL PNEUMONIA (Broncho-pneumonia : Lobular Pneumonia).

Definition: An inflammation of the lung, affecting the lobules, finer bronchi, and air-cells, usually following bronchitis, and in the great majority of cases due to infection.

Etiology: The micro-organisms most frequently found in catarrhal pneumonia are the micrococcus pneumoniæ crouposæ (Sternberg), Friedländer's bacillus, streptococcus pyogenes, staphylococcus pyogenes aureus, diphtheria bacillus, influenza bacillus, tubercle bacillus, and the typhoid bacillus. Mixed infection is very common.

Pneumonia due to the diphtheria bacillus, influenza bacillus, tubercle bacillus, and typhoid bacillus is treated of under Diphtheria, Influenza, Tuberculosis, and Typhoid Fever, respectively.

Frequently catarrhal pneumonia is due to the extension of a bronchitis. Capillary bronchitis, or bronchiolitis, rarely if ever exists except in the presence of pneumonia.

Since many of the micro-organisms found in pneumonia may be present in the respiratory tract, especially in the nose

and throat, and probably also in the lungs, in health, an important *rôle* in causation is ascribed to exposure to inclement weather, cold, and moisture, the inhalation of dust and anæsthetics, and trauma, which are believed to favor infection.

The so-called aspiration-pneumonia, which occurs most frequently after anæsthesia, is due to an invasion of the lung by micro-organisms, which gain access to the lung in abundance at the time of anæsthesia, through the increased secretion and diminished expectoration. In such cases infection may be favored by the irritation of the lung caused by the anæsthetic.

Symptomatology: The symptoms bear a general resemblance to those of croupous pneumonia, but show wide variations, as might be anticipated from the variety of agents that may enter into the etiology of catarrhal pneumonia. The symptoms of *carbonic-acid poisoning* and toxæmia assume importance.

The more important symptoms are *fever*, usually 102° to 103° F. in the evening, sometimes 104° to 105° F.; *rapid pulse*, 150 or higher, and respiration 20 to 60 or more; dyspnœa, and cough. The *pulse-respiration ratio* is altered from the normal 2 : 9 to 1 : 3–2 : 3.

Physical signs: *Percussion* may elicit some *dulness*, usually near the spine and low down. *Auscultation* reveals *râles* of various kinds. Bronchial breathing and bronchophony are the exception. Resolution may take place, but usually appears later than in croupous pneumonia.

The *duration* of the disease is longer in catarrhal pneumonia than in croupous pneumonia. *As a rule the temperature falls by lysis.* Often *convalescence* is protracted. Pericarditis, endocarditis, and meningitis occur only rarely.

Catarrhal pneumonia—diagnosis: The disease shows a preference for the extremes of life. The existence of some etiological factor, such as bronchitis, is often of value in diagnosis. Important symptoms are the elevation of temperature, increase of pulse and respiration, with disproportion of the pulse-respiration ratio, and the presence of *râles*.

Physical examination may show infiltration of parts of a number of lobes, involving usually both lungs. Croupous

pneumonia, on the other hand, shows consolidation of an entire lobe, and is usually unilateral.

In catarrhal pneumonia the sputum is muco-purulent and may contain blood, but is not of the rusty character found in croupous pneumonia. In some cases the microscopic examination of the sputum will reveal the true nature of the disease.

Prognosis: The mortality is much higher than in croupous pneumonia. The outlook is grave in children after measles, whooping-cough, and diphtheria. High temperature, with dyspnœa, irregular respiration, especially Cheyne-Stokes respiration, delirium, convulsions, and somnolence are ominous, especially late in the course of the disease.

Catarrhal pneumonia—treatment: The treatment is symptomatic. High fever, above 103° F., calls for hydrotherapy, best cold sponging, or a warm or cool bath. In the presence of a strong heart, especially in children, phenacetin or lactophenin, best given with whiskey or wine to avoid depression, may give considerable comfort. Such measures also address most pleasantly the nervous distress so often present. Pleuritic pain may be relieved by hot or cold applications, a mustard plaster, or the administration of opium. The patient should drink plenty of pure water, plain or carbonated, to which lemon-juice or cream of tartar may be added. Emetics are sometimes useful for the removal of the secretion from the trachea. Of more value are the stimulating expectorants, senega, ammonia, camphor, and benzoic acid.

A weak heart should be supported with cold sponging, digitalis, alcohol, strophanthus, caffeine, carbonate of ammonium. Nitroglycerin is of value in cases of arterio-scelerosis.

The patient should be kept upon a fever-diet: milk, soup, eggs; later oysters, chicken, and steak.

A change of climate may be necessary in chronic cases. In all cases the patient should receive an abundance of pure fresh air (see Treatment of Bronchitis).

Influenza-pneumonia (see Influenza): The prognosis is worse than in the other forms of catarrhal pneumonia or in croupous pneumonia.

Tubercular pneumonia (see Tuberculosis).

Typhoid pneumonia (see Typhoid Fever). A true typhoid pneumonia may be caused by the typhoid bacillus, and occurs especially in the course of typhoid fever, the infection of the lung probably occurring through the blood. In some cases the infection of the lung by the typhoid bacillus has seemed to occur through the respiratory tract (Richiardière). The fever, nervous symptoms, and course of the disease resemble those of typhoid fever. Sometimes the term "typhoid pneumonia" is applied incorrectly to a combination of typhoid fever and croupous or catarrhal pneumonia.

EMPHYSEMA (Pulmonary Emphysema).

Definition: *Vesicular emphysema* shows dilatation of the pulmonary alveoli with distention of the alveolar walls, which sometimes atrophy and disappear. In *interstitial emphysema* there is inflation of the interstitial lymph-spaces of the lung with air that escapes from the alveoli through rupture.

Etiology: The most prominent factor in causation is increased intrapulmonary pressure, due to expiratory effort. Emphysema is found most frequently in chronic bronchitis, especially in dry bronchitis and in certain occupations, among musicians who play on wind-instruments, glass-blowers, and those who do heavy lifting, in which the glottis is closed and the accessory expiratory muscles are brought into action. Congenital weakness of the pulmonary tissue seems to play a *rôle* in some cases.

Symptomatology: *Vesicular emphysema* comes on gradually and pursues a chronic course. With the disappearance of the alveolar walls the aërating surface in the lung is diminished. There is *dyspnœa*, which is *expiratory in character*, at first observed only on exercise, later becoming more constant. The dyspnœa is aggravated by bronchitis. Usually there is *cough*. Because of the lessening of the pulmonary vascular area the heart must do more work. *The right ventricle becomes hypertrophied, and later undergoes dilatation.* The tricuspid valve becomes relatively insufficient. There are *cyanosis and*

dropsy, which may become general to constitute a true anasarca. The *chest* comes to occupy the position of inspiration, —*becomes "barrel-shaped."* The chest appears as if the individual were holding his breath at full inspiration.

Inspiration is short and expiration prolonged and forced. The heart is pushed downward. The apex-beat may be in the sixth or seventh intercostal space. Epigastric pulsation is common.

Physical signs: *Percussion* reveals drum-like *tympanitic resonance; diminution, sometimes obliteration, of the cardiac dulness,* due to the heart being covered by the lung. The pulmonary resonance is increased downward, and the *liver may be pushed downward* so that it can be readily palpated.

Auscultation: Expiration is prolonged. In the presence of bronchitis, râles may be heard. Usually there is accentuation of the second pulmonary valve sound. With insufficiency of the tricuspid valve a systolic murmur is heard.

The liver and spleen may be enlarged, especially late in the disease.

Prognosis: In pronounced cases the prognosis is grave. Those who are able to take proper care of their health, especially with regard to the selection of climate and the treatment or prevention of bronchitis, may live for years in comparative comfort. Where this is not possible the duration of life is shortened. In all cases the disease runs a chronic course. Life often is terminated by some intercurrent malady. Otherwise death comes through failure of the heart.

Emphysema—treatment is symptomatic, and should be addressed especially to the prevention or cure of bronchitis and the support of the heart. The patient should reside in a warm climate during the winter. Where this is not possible the individual should remain in the house in winter and during inclement weather. The remedies of most value in the treatment of the bronchitis are iodide of potassium, citrate of potassium, and pilocarpine. Strychnine is an excellent tonic. A failing heart demands rest and the judicious use of digitalis and strophanthus. Œdema may be relieved by calomel or diuretin.

ATELECTASIS.

Definition: Collapse or incomplete distention of a greater or less number of pulmonary alveoli.

Etiology: Complete atelectasis is found normally in the lung of the fœtus. In the new-born it is evidence that the child has not breathed. Acquired atelectasis may be due to plugging of a bronchus or compression of the lung.

Symptomatology: Partial atelectasis shows *increase of respiration and absence of fever*, except when caused by associated processes. The respiration is superficial. The pulmonary area may be decreased and the cardiac area increased. The lung retracts from over the heart.

Percussion may reveal dulness or flatness over the affected portion of the lung.

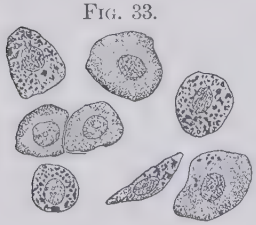
During life, atelectasis may be overshadowed by the symptoms of the disease or condition which causes it.

Treatment should address the cause, in the hope of prevention of complete atelectasis, which is incompatible with life.

ŒDEMA OF THE LUNG (Pulmonary Œdema).

Definition: A collection of fluid in the interstitial tissue of the alveoli and smaller bronchioles. The fluid comes from the blood, through the vessel-walls, and may be clear or tinged with blood.

FIG. 33.



Œdema pulmonum. Desquamated epithelial cells containing particles of coal-dust (Whittaker).

Etiology: Passive congestion of the lung, due to a weak heart, is probably the most common cause.

Œdema occurring in nephritis is due to changes in the vessel-walls or in the heart, weakness. Œdema may occur in the neighborhood of inflammatory processes in the lung.

Symptomatology: The onset may be sudden or gradual. There are *dyspnœa, cyanosis, and increased frequency of respiration.* *Râles* occur, at first with resonance, later with dulness or flatness over the more dependent portions of the lung.

The *sputum* contains œdematous cells, known as cells of pulmonary œdema or the cells of heart-failure (Fig. 33). The sputum may contain urea in cases occurring in nephritis.

The **prognosis** is always grave, but depends upon the cause, especially upon the reaction of the heart to stimulation.

Pulmonary œdema—treatment: Most cases are due to a weak heart, which should be strengthened by rest and the judicious use of cardiac stimulants and exercise. Grave cases may call for the analeptics, probably best, camphor and oil, 1 : 8, hypodermatically. Often considerable comfort is secured by the use of morphine.

ABSCESS OF THE LUNG.

Abscess of the lung may be single or multiple.

Etiology: Among the causes of abscess of the lung are: tuberculosis, pneumonia, empyema, mediastinitis, œsophageal carcinoma, abscess of the liver, subdiaphragmatic abscess, embolism, and the presence of a foreign body in the lung.

The **diagnosis** is difficult in the absence of expectoration of pus. Sometimes pus may be detected by aspiration. In the differentiation from a bronchiectatic cavity, when pus is expectorated, the finding of portions of the lung or elastic tissue would speak for abscess.

Treatment: If possible, the abscess should be treated surgically, opened and drained. In other cases the treatment must be expectant, symptomatic. As far as possible the cause should be addressed.

GANGRENE OF THE LUNG.

Primary gangrene of the lung, due to trauma, is rare. Gangrene of the lung is most frequently caused by pneumonia, infarction, embolism, abscess, echinococcus, actinomycosis, neoplasms; rarely by tuberculosis. The disease shows a preference for males, poverty, and the age of twenty to fifty years.

Symptomatology: The *odor of the breath* is very offensive. *Expectoration* is usually abundant, and the *sputum* is foul-smelling, and upon standing separates into three layers: the

upper muco-purulent, the middle thin and watery, and the lower purulent. *Microscopical examination* of the sputum reveals pieces of lung-tissue, especially elastic fibres, numerous bacteria, mould-fungi, and both fat-crystals and free fat. If the gangrene involve a considerable area, it may be recognized by the presence of *dulness and bronchial respiration;* or, in the presence of a *cavity*, by tympanitic resonance, especially the *cracked-pot sound*, and *amphoric respiration.*

The **prognosis** depends largely upon the cause and the strength of the patient, but is always grave. Death is usually caused by exhaustion, sometimes by hemorrhage, rarely by abscess of some other organ, especially of the brain.

Treatment: Inhalations of creosote lessen the offensive odor of the breath. Rarely surgery may benefit a case by incision and drainage. As a rule the treatment is purely symptomatic.

PNEUMONOKONIOSIS.

Definition: Disease due to the *inhalation of dust.*

Varieties: *Anthracosis* or anthraco-pneumonokoniosis, coal-miners' phthisis, coal-miners' lung, due to the inhalation of coal-dust. *Siderosis*, knife-grinders' phthisis, refers especially to disease caused by the inhalation of particles of metal (steel and iron). *Chalicosis* is due to the inhalation of mineral dust. *Millers' phthisis* is due to the inhalation of particles of wheat, especially of the hull of the grain.

The **symptoms** are those of bronchitis, emphysema, interstitial pneumonia, or tuberculosis.

The **diagnosis** rests upon the symptoms and occupation of the patient, and the character of the sputum, which contains particles of the dust inhaled. Frequently the irritation caused by the dust opens the way for invasion by micro-organisms. Many of the patients succumb to tuberculosis.

Prognosis: Mild cases recover upon a change of occupation. In advanced cases the prognosis is bad. The invasion by micro-organisms, especially by the tubercle bacillus, adds gravity to the prognosis.

Treatment: Something may be done in the way of prophy-

laxis by the use of inhalers, or of apparatus to remove the dust, especially in factories.

The treatment of a case calls for a change of occupation. Further treatment is that of bronchitis.

Syphilis of the lung (see Syphilis).

Echinococcus of the lung (see Echinococcus).

Actinomycosis of the lung (see Actinomycosis).

DISEASES OF THE PLEURA.

PLEURISY.

Definition: An inflammation (infection), acute or chronic, of the membrane lining the pleural cavity.

Etiology: Tuberculosis is the most frequent cause. Some cases are due to pneumonia, infarctions, rheumatism, syphilis, and infection with the typhoid bacillus. Typhoid infection of the pleura may occur either with or without intestinal lesions. Charrin and Roger (1891) found infection of the pleura with the typhoid bacillus, without infection of the intestine, in a postmortem upon a case in which there were the symptoms of typhoid fever, except those symptoms due to lesion of the intestine. Pleurisy may be caused by trauma or by Bright's disease. The streptococcus pyogenes is found most frequently in purulent pleurisy (see Empyema). Exposure to cold has come to occupy a subordinate place in etiology.

Pleurisy—symptomatology: The acute attack comes on suddenly with *chill followed by fever*, 102° to 103° F., and *increased pulse-rate*. As a rule the most prominent early symptom is *pain*, usually in the side, which is aggravated by pressure, cough, or deep inspiration. At first the patient lies on the well side, to avoid pain, and later on the affected side, to secure greater freedom of respiration. *Effusion* probably begins soon after the onset of the disease, but usually may not be readily detected until the second to the fifth day. *With the separation of the pleural surfaces by the effusion the*

pain disappears. Usually the pulse and respiration are increased in frequency. As a rule the temperature continues high. *Dyspnœa* may be troublesome.

Sometimes the *onset* of pleurisy is *insidious.* The patient complains of cough and shortness of breath brought on or increased by exercise. There may be pain in the side. The general health is impaired, the appetite is poor, and there is weakness, frequently pallor. Such cases occur most frequently at the extremes of life, usually secondary to other diseases, especially tuberculosis and chronic diseases of the heart and kidneys.

In some cases there is no effusion, constituting the so-called *dry pleurisy.* Effusion, when present, may last two to five days in rheumatic cases (Netter); usually four to six weeks in acute cases with small or moderate effusion; and a number of years in chronic cases, before absorption takes place.

Pleurisy—physical signs: At first the most important sign is the *pleural friction-rub*, heard both upon inspiration and expiration. Later there is the *evidence of effusion*, appearing first as *dulness* over the most dependent portion of the pleural cavity. The friction-sound may still be heard above the area of dulness. With increased effusion the dulness becomes more pronounced; there is absolute flatness. *The respiratory movement of the affected side is diminished. Vocal fremitus is absent* over the effusion and increased over the compressed lung. *Large effusion causes distention* of the affected side, displacement of organs, and bulging of the intercostal spaces. In marked cases auscultation may detect no sounds upon the affected side. Usually there are *bronchial breathing and bronchophony*, occasionally ægophony. After absorption there is a *return of the friction-sound*, which is found over a larger area than at first. There are numerous crackling râles.

The early **diagnosis** of pleurisy or the detection of mild cases depends largely upon the recognition of the friction-sound. Later, dulness and flatness are characteristic. Attention often is first directed to the chest by the pain in the side. The symptoms may indicate the character of the exudate, which can be determined positively only by puncture. The

examination of the exudate, microscopically and by inoculation and culture, may reveal the micro-organisms present in a given case.

The **prognosis** depends largely upon the cause. So-called rheumatic pleurisy almost invariably pursues a short and favorable course. Many cases of pleurisy seem to recover from the attack, and succumb later to tuberculosis. But even tubercular pleurisy may recover. Chronic pleurisy may cause permanent deformity of the chest.

Pleurisy—treatment: The patient should remain in bed. Pain may be relieved by hot applications and poultices, and by strapping the side to prevent the movements of respiration. Severe pain calls for opium, best in the form of Dover's powder in broken doses; or morphine hypodermatically. The bowels must be kept open. Fever is relieved best by cold sponging. Pleurisy due to tuberculosis, rheumatism, or syphilis should be treated with remedies addressed to these diseases—tuberculin, the salicylates, and iodides, respectively.

An exudation that threatens life or is very slowly absorbed must be removed by aspiration or incision. Symptoms demanding aspiration are asphyxia, weakness of the heart, rising of the fluid to or above the third interspace with the patient in the erect position, and delayed absorption.

The *puncture* is best made in the fourth interspace on the left side, or the fifth interspace on the right side. Not all the fluid should be removed.

EMPYEMA (Purulent or Suppurative Pleurisy).

Etiology: The most frequent causes are the streptococcus pyogenes and the micrococcus pneumoniæ crouposæ. The tubercle bacillus opens the way for invasion of the pleura by other micro-organisms. The staphylococcus is usually found associated with the tubercle bacillus or the micrococcus pneumoniæ crouposæ. The bacillus of Friedländer, the typhoid bacillus, and saprophytic micro-organisms are sometimes present.

Infection of the pleura may come from the *lung*, from pneumonia, tuberculosis, abscess, gangrene, infarction, bronchiec-

tasis, and cancer; from the *chest-wall*, from inflammations of the skin, lymphatic glands, or breast, especially cancer of the breast and peripleuritis; from the *mediastinum*, from mediastinal abscess, pericarditis, and cancer of the œsophagus; from the *abdomen*, from peritonitis, and hepatic, subdiaphragmatic, and perityphlitic abscess; and from certain *infectious diseases*, especially septicæmia (puerperal fever), erysipelas, influenza, scarlet fever, and diphtheria. Infection may occur through trauma (wounds).

Symptomatology: Aside from the *symptoms of pleurisy*, in *empyema* there is evidence of the *presence of pus* in the pleural cavity. The *onset* of empyema may be sudden or insidious, and the course of the disease may be acute or chronic. Usually sooner or later the temperature shows the curve characteristic of *septicæmia*. At the same time there are *emaciation* and *loss of strength*. The *dyspnœa* becomes greater than may be accounted for by the amount of fluid present in the pleural cavity.

The symptoms vary somewhat with the cause. In empyema due to the *streptococcus pyogenes* the streptococcus (hectic) temperature-curve is usually present from the beginning. To this class belong, as a rule, the fulminant cases. Œdema of the chest-wall is frequent. Often there is enlargement of the axillary glands. Exceptionally metastatic abscesses occur, most frequently in the brain.

Empyema due to the *micrococcus pneumoniæ crouposæ* is usually, if not always, secondary to pneumonia, which may pass unrecognized. Frequently the course resembles that of pneumonia, with sudden onset, pain in the side, cough, and marked improvement in seven to nine days. Cases occurring after the crisis of pneumonia show a less characteristic course. Œdema of the chest-wall is rare. Spontaneous evacuation, oftenest through the lungs, less frequently through the intercostal spaces, occurs in at least one-fourth of the cases.

Encapsulation of the exudate occurs more frequently than in other varieties of empyema. The pus is dense and viscid, usually of a grayish-yellow color. This form of empyema terminates, as a rule, in recovery, which may account for the usually favorable course of empyema in children.

Cases of empyema in which the *tubercle bacillus* is present are usually insidious in onset and pursue a chronic course.

Fœtid or putrid empyema is caused by the presence of *saprophytic micro-organisms*. Frequently *gangrene* of the lung is the source of the infection. The cases usually present marked symptoms of sepsis. Often the expectoration is offensive, even in the absence of discharge of the empyema.

Cases of empyema due to the *typhoid bacillus* usually terminate favorably. As a rule, the fever resembles that of typhoid fever.

In many cases there is *mixed infection*.

Diagnosis: The symptoms of pleurisy in combination with the evidence of septicæmia may lead to the suspicion of empyema. Œdema of the chest-wall occurring in pleurisy would indicate empyema. A positive diagnosis may be made by the withdrawal of pus, through aspiration or incision.

Prognosis is good, provided early evacuation of the pus is obtained. Many cases undergo resolution without operation. Empyema is, however, a serious disease. Much depends upon the cause.

Treatment: The pus may be absorbed, especially in *children*. As long as the general condition of the patient remains good, in the absence of marked evidence of septicæmia, the treatment may be expectant.

Impairment of the health of the individual, especially changes in the pulse and respiration, calls for surgical interference, incision, and drainage. Sometimes it is necessary to resect part of a rib to secure thorough drainage of the empyema.

HYDROTHORAX.

Definition: An œdematous transudation of fluid into the pleural cavity.

Etiology: The causes are those which may produce œdema elsewhere: obstruction to the circulation, due to disease of the heart or of the lung (emphysema); hydræmia, due to kidney disease or cachexia.

Symptomatology: There is *dyspnœa*, which may be aggra-

vated by the conditions that produce the hydrothorax. Hydrothorax does not cause pain or fever.

Physical examination reveals *fluid in the pleural cavity*, the character of which may be determined by aspiration. Hydrothorax is almost always bilateral.

Diagnosis: The occurrence of bilateral transudation of fluid into the pleural cavity, in the presence of general œdema, is characteristic. Doubtful cases may be cleared up by aspiration.

The **prognosis** depends upon the cause.

Treatment should address the cause. In bad cases the fluid may be withdrawn by aspiration. In the presence of general dropsy, relief may be obtained by increasing the action of the heart, kidneys, and bowels; or fluid may be withdrawn from the legs by the introduction of silver canulæ into the subcutaneous tissue.

PNEUMOTHORAX.

Definition: Air in the pleural cavity. A combination of pneumothorax and hydrothorax constitutes *pneumo-hydrothorax*. The presence of air and pus in the thorax is known as *pneumo-pyo-thorax*.

Etiology: Air may gain access to the pleural cavity through *perforation*. Cases may rarely be due to the presence of anaërobic *gas-forming micro-organisms*. Such organisms were found by Levy in a case of pneumothorax following pleurisy. But perforation is the more common cause.

The perforation may be caused by trauma, as by a broken rib or rupture of the lung. Aside from trauma, the majority of cases are due to tuberculosis. Other causes are empyema, emphysema, pneumonia, gangrene of the lung, abscess of the lung or liver, carcinoma, and the emptying of a bronchiectatic cavity into the pleural cavity.

Pneumothorax—symptomatology: Usually the *onset is sudden*, with *pain*, *dyspnœa*, and *cyanosis*. There may be cough. *Prostration* is marked. The *pulse and respiration are increased*, *the temperature subnormal*. The patient may pass into collapse and die within a few hours or days; or death may occur

later from exhaustion. In other cases the symptoms improve and recovery follows with absorption of the air or gas.

Physical examination shows enlargement of the affected side, with displacement of the organs,—heart, liver, and spleen,—as in pleurisy. Vocal fremitus is diminished or absent over the affected area and increased over the collapsed lung. The percussion-note may be tympanitic, but is usually only loud with a low pitch. At any rate, the percussion-note over the affected area differs from that over the normal lung. Auscultation over the affected area reveals diminution of the respiratory murmur. The sounds of respiration and the voice are distinctly amphoric (metallic tinkle, which may also originate in the stomach).

The presence of fluid or pus (pneumo-hydro-thorax, pneumo-pyo-thorax) may give rise to *succussion*. This should not be mistaken for succussion occurring in the stomach. Succussion may also occur in a large cavity in the lung. Soon the symptoms of fluid in the pleural cavity appear (see Pleurisy). The fluid changes its level with changes in the position of the body more readily than when the hydrothorax is not accompanied by the presence of air or gas. Usually the air or gas is soon absorbed after the appearance of fluid.

Diagnosis: The sudden onset, dyspnœa, and the physical signs, especially the increased resonance, with feeble or amphoric respiration over the affected area, and the displacement of organs, especially of the heart and diaphragm (liver and spleen), are characteristic. The respiratory sounds may be entirely absent. Light cases are sometimes difficult to diagnosticate.

Differential diagnosis has to do chiefly with emphysema, pulmonary cavities, hernia of the diaphragm, and pyo-pneumothorax subphrenicus.

Prognosis: In the absence of infection the prognosis is good. Tubercular cases have a worse outlook. The occurrence of pus (pneumo-pyo-thorax) adds gravity to the prognosis. The prognosis is unfavorable in double pneumothorax.

Pneumothorax—treatment: Pain should be relieved with hot applications, poultices, or opium, preferably in the form of morphine hypodermatically. Prostration and collapse

should be met with the analeptics, alcohol, sodium benzoate, of caffeine, camphor, ether, digitalis, strychnine. Asphyxia may necessitate puncture with a hypodermatic needle or fine trocar, best made in the fourth to sixth interspace in front.

Later, effusion may call for the intervention of surgery, aspiration, or incision and drainage.

Echinococcus of the pleura (see Echinococcus).

Malignant diseases of the pleura: *Sarcoma* rarely invades the pleura.

Carcinoma of the pleura is almost always secondary to carcinoma elsewhere. Primary carcinoma of the pleura has been reported in a few cases.

CHAPTER IV.

DISEASES OF THE ORGANS OF CIRCULATION.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition: An acute or chronic inflammation of the pericardium.

Etiology: *Primary pericarditis* may be due to trauma or causes apparently not connected with other disease, such as "taking cold."

More important, because much more frequent, are the cases of *secondary pericarditis*, which may be caused by the infections or by extension of inflammation from contiguous organs, due to bacterial invasion or the action of toxins. Pericarditis is most frequently found in rheumatism, especially in acute articular rheumatism, chorea, tuberculosis, pleurisy, endocarditis and myocarditis, pneumonia, influenza, scarlatina, septicæmia, variola, scorbutus, nephritis, gout, cholera, dysentery, erysipelas, diphtheria, cerebro-spinal meningitis, hæmophilia, hemorrhagic diathesis, purpura, morbus maculosus, leukæmia, diabetes, cirrhosis of the liver, carcinoma, sarcoma, and syphilis; typhus, typhoid fever, intermittent fever, relapsing fever, gonorrhœa, phlebitis, and osteomalacia. Aneurism is a rare cause.

Symptomatology: The symptoms of pericarditis may be slight, overshadowed by associated disease, or entirely absent; again, they are pronounced.

Sometimes the onset is sudden, with chill and rigor, a rise of temperature, malaise, anorexia, headache, and dizziness. There may be palpitation of the heart. In other cases the onset is insidious and these symptoms are not present. Some-

times, especially in the *aged*, the temperature may be *subnormal*.

Frequently pericarditis first manifests itself by *pain*, which sometimes extends to the left shoulder and down the arm. There may be tenderness over the region of the heart and in the epigastrium. Sometimes the pain is increased by inspiration. Later, upon the appearance of *effusion*, the pain disappears or at least is diminished. The effusion interferes with the action of the heart. The pulse becomes weak and irregular. Exertion or excitement may be followed by *syncope*. Frequently there is *dyspnœa*, which may amount to *orthopnœa*, with *cyanosis*. Interference with the heart's action may lead to *œdema*, especially of the extremities, in some cases assuming the proportions of an anasarca.

There may be distention of the cervical veins, with venous pulsation, dysphagia, and cough, sometimes aphonia, from pressure on the recurrent laryngeal nerve.

Usually the patient lies upon the back, in a semirecumbent posture. The urine is high colored, and may contain albumin and blood, rarely casts.

Pericarditis—physical signs: *Inspection* may reveal *distention of the ribs, especially in children*, and the presence of considerable effusion. There may be more or less *restriction of the respiratory movements*. Sometimes a large effusion produces only *widening and bulging of the intercostal spaces*, because of the inelasticity of the ribs. The *apex-beat* may be displaced to the left and upward. With absorption of the effusion all these signs disappear.

Palpation may or may not detect *tenderness* over the heart or in the epigastrium. A friction-*fremitus* may be felt. There is dislocation of the *apex-beat*, which may change with the position of the body. *Effusion*, causing great distention, may be recognized by palpation. After absorption of the effusion the friction-fremitus may again become perceptible, and the apex-beat resumes its normal position. The friction-sound may disappear from effusion, adhesion, or resolution.

Percussion shows enlargement of the *heart-dulness*, when there is any considerable amount of effusion. *Dulness in the fifth intercostal space to the right of the sternum* occurs early

in pericardial effusion. In extreme cases the dulness may extend from the second rib, sometimes as high as the clavicle, down to the ensiform appendix, and from nipple to nipple.

Auscultation is of most value in early diagnosis. *Pericardial friction-sounds*, varying in character, occur synchronous with the heart-sound, sometimes with respiration. The friction-sounds are heard best during full inspiration with the body inclined forward, and are increased by pressure over the heart. The friction-sounds become less distinct, and finally disappear with the occurrence of *effusion*. *The heart-sounds become muffled.* Upon absorption of the effusion a friction-sound may again be heard, and the heart-sounds again become normal. The friction-sounds finally entirely disappear with absorption of the fibrin or adhesion of the pericardial surfaces.

Diagnosis: The symptoms may be suggestive, but a diagnosis can be made only upon physical examination. The pericardial friction-sound and the evidence of effusion, especially dulness in the fifth intercostal space on the right of the sternum, the precordial dulness later assuming the shape of the pericadial sac, with the base of the triangle above, are characteristic. Aspiration may be necessary to detect effusion, and at the same time will reveal the character of the effusion. Sometimes aspiration may not detect fluid in the pericardium even when present.

Differentiation concerns especially endocarditis, pleurisy, hypertrophy of the heart, mediastinal tumors, and irritation or inflammation of the stomach.

The **prognosis** varies with the cause, extent, and character of the inflammation and the general condition of the patient, especially the strength of the *heart-muscle*. The mortality is high at the extremes of life. The outlook is bad in tubercular or purulent pericarditis. In all cases the prognosis should be guarded. Usually the cases due to rheumatism are lighter than those due to Bright's disease, pyæmia, or scurvy.

Pericarditis—treatment: The first requisite is absolute rest in bed. Cold applications—an ice-bag or Leiter's coil—may be used early. Sometimes these are not tolerated, when they may be substituted by hot applications. It is better to keep

the patient on a fever-diet; milk and eggs form the best food. Opium, best in the form of Dover's powder, or morphine, may be given to relieve pain and quiet the heart's action. Violent action of the heart is best relieved by rest and the application of cold. Temperature that is excessive may be controlled by sponging with cold water.

In the treatment of pericarditis *due to rheumatism* the salicylates are advised. DaCosta believes them useless, and that they may do harm by depressing the heart.

Weak and irregular action of the heart may be met with digitalis.

Large effusions may demand *paracentesis*, best in the fifth interspace about two inches to the left of the median line. Potassium salts, especially the iodide, acetate, and citrate, best in combination with digitalis, have been recommended to promote absorption of the effusion. In the presence of fever and irregular pulse quinine may be administered, gr. iij–v every four hours.

Purulent cases should be treated surgically, by incision and drainage.

PERICARDIAL EFFUSIONS.

Effusions into the pericardial cavity, according to their character, are known as *hydropericardium*, or hydrops pericardii, clear fluid in the pericardium; *hæmopericardium*, blood in the pericardium; *pyopericardium*, pus in the pericardium; and *pneumopericardium*, when there is air in the pericardium.

DISEASES OF THE HEART.

It is now believed that both contraction and dilatation of the arteries and heart are active processes. In embryonic life an aggregation of cells takes place in the middle germinal layer, which forms a network in the area pellucida. Within these cells, cavities develop, the primary capillaries, from which there are offshoots, the secondary vessels, which traverse the body as bloodvessels. The heart, which has been aptly described as a quadruplication of the bloodvessels, is developed later.

ATROPHY OF THE HEART.

Atrophy of the heart may be partial, involving only a part of the heart; or complete, involving the whole heart.

Etiology: Sometimes the condition is congenital. A partial atrophy may be due to chronic endopericarditis. Usually acquired atrophy of the heart is general, associated with general wasting of the body. Thus we find atrophy of the heart in the marasmus of phthisis, cancer, diabetes, amyloid degeneration of the kidneys, etc. The walls of the heart may show atrophy from arteriosclerosis.

Atrophy—symptomatology: The *heart's action* becomes weakened, the pulse feeble, the *impulse* of the heart diminished. Usually other evidences of *marasmus* are present. The area of *heart-dulness* is diminished. With weakness of the heart muscle the first sound becomes muffled and may not be heard; the second sound may be accentuated.

Diagnosis: Is made by the marasmus, atrophy of other organs, weak heart-action, and diminution of the area of heart-dulness.

The **prognosis** takes color with the cause. The immediate outlook depends upon the condition of the heart.

Atrophy—treatment: This should address the cause. A flagging heart calls for the judicious use of heart-stimulants. Probably one of the best is strychnine. Above all, the individual should live a pleasant life in an abundance of fresh air and sunshine.

HYPERTROPHY AND DILATATION OF THE HEART.

Cases of hypertrophy of the heart may be divided into (1) hypertrophy caused by some *obstruction within the heart*, especially valvular disease; and (2) so-called "idiopathic" hypertrophies, the cause of which may be: (*a*) disease of the heart-muscle, especially infection, over-strain and degeneration; (*b*) some obstruction in the vascular system outside of the heart, especially arteriosclerosis; and (*c*) affections of the nervous system.

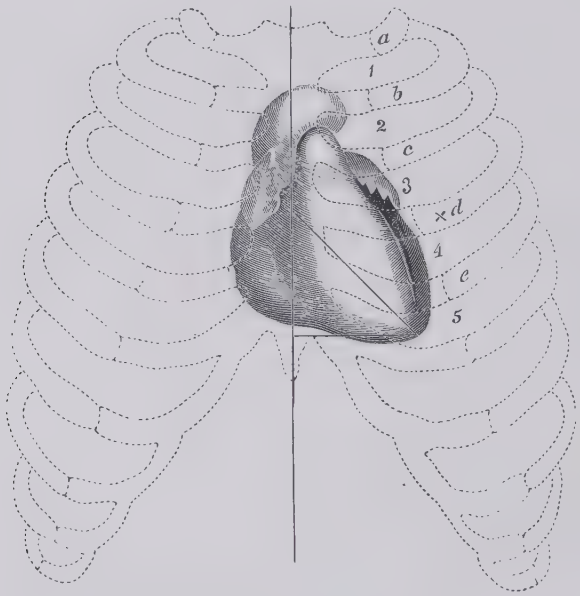
In some cases the cause may not be found, when the condi-

tion may be properly classed as a *cryptogenetic hypertrophy.* Hypertrophy is caused by some obstruction to the circulation, and is compensatory so long as it overcomes the obstacle.

Diseases of the heart-muscle which may cause hypertrophy and dilatation of the heart are: (1) fatty degeneration; (2) myocarditis; (3) tumors of the heart (myomata, cysts, malignant growths); and (4) parasites (cysticerci, echinococci).

Resistance within the vascular system, which may cause hypertrophy and dilatation of the heart, may be due to: (1)

FIG. 34.



Superficial cardiac dulness (approximate) (Flint).

congenital contraction of the vessels; (2) arteriosclerosis; (3) muscular effort (hard work, strain); (4) plethora (excess in eating and drinking); (5) pregnancy; (6) disease of the kid-

ney (Bright's disease); (7) diseases and deformities of the chest (emphysema, kyphosis).

Among the **affections of the nervous system** that may cause hypertrophy and dilatation of the heart are: (1) mechanical irritation of the vagus nerve (tumors, enlarged lymphatic glands, Basedow's disease); (2) chemical irritation of the vagus (alcohol, tobacco, coffee, tea); (3) psychic irritation (domestic troubles, business worries); (4) excess in venery.

Symptomatology: *Hypertrophy of the heart* is indicated by increased *pulse-tension*, dislocation of the *apex-beat* to the left, increase of the *impact of the heart and dulness*, accentuation of the second aortic valve sound. The increased blood-supply may cause headache, epistaxis, and polyuria.

Dilatation of the heart is indicated by the signs of a failing heart, *frequent and small pulse*, *dyspnœa*, *bronchitis*, *asthma*, *headache* (caused by anæmia of the brain), and *œdema*, first of the ankles and eyelids, sometimes of the lungs, which may finally amount to anasarca.

Dilatation may cause a valve to become insufficient in the absence of any disease of the valve itself. Such *relative insufficiency* or incompetency is most frequently found in the tricuspid valves, due to dilatation of the right ventricular orifice, most frequently the result of mitral regurgitation. In such cases a bruit may be heard over the ensiform cartilage.

Diagnosis: The symptoms of most value in the diagnosis of *hypertrophy of the heart* are: (1) the dislocation of the apex to the left; (2) accentuation of the second aortic valve sound; (3) increased cardiac impact and dulness; (4) the full pulse; and (5) evidence of increased blood-pressure in various organs (headache, epistaxis, polyuria).

Dilatation of the heart is recognized by the weakened heart's action, frequency of the pulse, tachycardia, palpitation, dyspnœa upon slight exertion, reduction in the quantity of urine, with usually enlargement of the liver, later œdema. Soon the urine assumes the character of the urine of stasis—high colored, containing hyaline casts.

Most important is the discovery of the cause: syphilis, alcohol, Bright's disease, tobacco, coffee, tea, valvular disease, etc.

The *differential diagnosis* concerns especially (1) pericarditis, in which the area of dulness takes the form of the pericardial sac, with the base upward; the impact of the heart and the apex-beat are weak and sometimes may not be felt upon palpation; and there are the characteristic friction-sound, fever, pain, etc.; and (2) aneurism of the aorta, in which there is the aneurismal bruit or thrill. The heart may be hypertrophied in cases of aneurism of the aorta.

The **prognosis** depends upon (1) the condition of the heart. In *compensatory hypertrophy* the prognosis is good. The outlook becomes bad when the hypertrophy gives way to dilatation.

(2) The prognosis depends largely upon the *cause*. The prognosis is good in the hypertrophy of pregnancy; but pregnancy may make the prognosis worse when it occurs in the course of disease of the heart. The outlook is not bad in hypertrophy due to excess in venery, or the abuse of tobacco, tea, or coffee, if such excess or abuse be discontinued. The prognosis is more grave in arterio-sclerosis and Bright's disease.

(3) The prognosis takes color with the occurrence of certain symptoms: uræmia, cyanosis, and tachycardia are grave signs.

(4) The outlook is bad when the heart no longer responds to digitalis or other heart-stimulants.

Prophylaxis: Care should be exercised not to break down an hypertrophy that is compensatory. This calls for control of the emotions, especially worry, anger, and fear; and avoidance of excesses, strains, and exposures.

Treatment: Tumultuous action of the heart may be controlled by rest and cold applications (cold compresses, ice-bag) over the heart, and the administration of the bromides, sodium bromide, gr. xx–xl in a glass of water, or Selters water.

The symptoms of *dilatation* call for rest, best in bed in the recumbent posture, whereby the greatest relief is obtained from dyspnœa, palpitation, and heart-failure.

Upon recovery of the tone of the heart, marked by improvement in the pulse and an increased secretion of urine, judicious exercise of the heart-muscle may be recommended.

Such exercise is secured by very gradual increasing gymnastic exercise, best in the open air, but never continued to the point of fatigue. Contraindications to such exercise are acute myocarditis and acute dilatation of the heart.

The diet should be light and nutritious, and may consist largely of milk.

Compensatory hypertrophy should not be interfered with by the administration of heart-stimulants. With the breaking of compensation heart-stimulants become useful. Digitalis stands first. In mild cases the tincture of digitalis, gtt. v–x, may be given every three or four hours; in more severe cases the infusion of digitalis, teaspoonful to a tablespoonful at the same intervals. Digitalis is probably more effective in the form of the powder, a grain every two to six hours. Sooner or later digitalis disturbs the stomach, when it may be substituted by the tincture of strophanthus, gtt. v–x every three or four hours, or the sulphate of sparteine, in the same dose as the sulphate of morphine. The heart may be supported for a long time by strychnine, in the form of the tincture of nux vomica, gtt. x–xx, or of the sulphate or nitrate of strychnine, gtt. x of a grain-to-the-ounce solution three times a day. The sodium benzoate of caffeine, gr. iij–v, especially when injected subcutaneously, acts more readily. Nitroglycerin is especially valuable in the cases due to kidney disease (see Treatment of Heart-failure).

Further treatment is symptomatic.

MYOCARDITIS.

Inflammation of the myocardium, heart-muscle, may be acute or chronic; circumscribed or diffused; parenchymatous, involving chiefly the muscular fibres; or interstitial, involving chiefly the interstitial tissue.

Etiology: *Circumscribed myocarditis* may be caused by embolism in the coronary artery or its branches, or by septicæmia. The disease is often associated with ulcerative endocarditis, puerperal fever, malignant pustule, acute articular rheumatism, diphtheria, or typhoid fever, and with purulent or gangrenous affection of the lungs.

Acute diffuse myocarditis, whether parenchymatous or interstitial, usually is caused by the infections, especially septicæmia, typhoid fever, diphtheria, pneumonia, and gonorrhœa.

Chronic myocarditis also may depend upon the infections, especially acute articular rheumatism, malaria, syphilis, gout, diabetes, Bright's disease. Many cases are caused by alcohol, tobacco, and lead. Probably most cases are ascribed to cold, trauma, or strain. Myocarditis is frequently due to the extension of inflammation from the endocardium or pericardium.

Symptomatology: Symptoms of myocarditis may be absent or overshadowed by endocarditis or pericarditis. As a rule, the heart is not able to do its work so well. Slight exertion causes *palpitation* and *shortness of breath*. There may be *pain* in the region of the heart, extending to the right arm or epigastric region. The apex-beat, impact of the heart, and the heart-sounds are weakened, indicating a *weak heart*. The *pulse* becomes *weak* and *irregular*. Frequently the *respiratory passages* show *catarrh*. *Digestion* is impaired. All the organs suffer from the poor blood-supply. The individual is *cyanotic*. The veins of the neck are distended.

The **diagnosis** rests chiefly on the evidence of a weak heart and the history of some disease that may play a *rôle* in etiology.

The **prognosis** is always grave, but recovery is the rule. The outlook is better in typhoid fever than in scarlet fever or diphtheria. The occurrence of pericarditis or endocarditis adds to the gravity of a case. The prognosis is bad in Bright's disease. Failure of digitalis is a bad sign.

Prophylaxis: The patient should take very carefully graduated exercise during convalescence from the infections—diphtheria, typhoid fever, etc. Excesses in gymnastics, venery, drinking (especially alcohol), and eating should be avoided. As far as possible the infections and obesity should be prevented. The proper treatment of the infections will do much to lessen the number of cases of myocarditis.

Treatment: The patient with *acute myocarditis* should observe absolute rest in bed, and not arise from the recumbent posture under any pretext. This implies the use of the bed-

pan, and not permitting the patient to sit up when being examined. The best single article of diet is milk, to which may be added fruit, fish, and the white meat of fowl. The supper should always be light, consisting only of a glass of warm water or weak tea.

In *chronic myocarditis* the patient should take carefully graded exercise, best in the open air. Such exercise delays or stops degeneration of the muscular fibres of the heart. Sometimes a change of climate is advisable. Bad cases may prefer the seashore, especially during the summer. It may be better to go to a warmer climate during the cold months. With improvement or with the beginning of treatment in light cases the heart may be given additional exercise by altitude, which at first should not be too high.

Cases of emphysema, asthma, and chronic bronchitis should lead neither a too active nor a too indolent life.

The *effect of toxins upon the heart* is met by quinine, gr. iij every three or four hours. The heart should be supported by strychnine, best hypodermatically. Further support of the heart calls for digitalis and the treatment given under Heart-failure. The sulphate of sparteine, gr. $\frac{1}{4}$, may be given hypodermatically several times a day; or camphor dissolved in oil or ether, hypodermatically; or the sodium benzoate of caffeine, internally or hypodermatically. Friction of the skin, especially of the extremities, hot applications, and mustard-plasters address the heart. The hot bath may be used when the heart is weak; in other cases the use of the cold bath daily gives relief to the patient and strengthens the heart.

ENDOCARDITIS (Acute Simple Endocarditis; Septic Endocarditis).

Etiology: Endocarditis is a secondary process, occurring in the course of or following some infectious disease, due to the invasion of the endocardium, or lining membrane of the heart, by micro-organisms, a number of which have been demonstrated, among them the micrococcus pneumoniæ crouposæ, streptococcus pyogenes, staphylococcus pyogenes

aureus, bacillus diphtheriæ, the gonococcus, and the tubercle bacillus.

In experiments upon animals it has been shown that the injection of micro organisms into the circulation is not followed by endocarditis unless the heart is first subjected to traumatic or chemical injury. This would seem to explain the *rôle* played by trauma, exposure to cold, arteriosclerosis, and atheroma.

Endocarditis is especially likely to appear in the course of, or after, rheumatism, pneumonia, influenza, septicæmia, including surgical sepsis and puerperal fever; also osteomyelitis, periostitis, erysipelas, furunculosis, and dysentery, gonorrhœa, scarlet fever; less frequently smallpox, measles, typhoid fever, syphilis, Bright's disease, and malaria. Sometimes even trivial affections (quinsy, mumps) may be accompanied or followed by endocarditis. The endocarditis may be due to invasion by the specific micro organisms of the infectious diseases, or to secondary infection, or to the effect of toxins.

Acute endocarditis—symptomatology: As a rule the *onset is insidious*, and the disease may remain unrecognized for a long time. As long as the inflammation is limited to the wall of the heart there may be no signs of the disease appreciable during life. Affection of the valves may occur without signs. Usually the valves are affected and the signs of the disease present; but frequently they are overlooked through failure to examine the heart.

Exceptionally endocarditis is announced suddenly by severe pain in the heart, dyspnœa, and cyanosis, rapid and irregular action of the heart. Such symptoms may follow sudden strain, with rupture of a diseased valve.

Suspicion may be aroused by *irregular fever*. But in some cases there is no fever. *The pulse is irregular and rapid.* There is *palpitation*, especially upon exercise or emotion. *Headache, insomnia, and anorexia* are common symptoms. The *spleen* is enlarged, hard, and tender. There may be enlargement and tenderness of the *liver*, such as occur in the infections. There is tenderness upon palpation over the region of the *kidneys*. The *urine* is high-colored, and may contain blood, albumin, casts, and micro organisms.

The detachment of thrombi may give rise to *embolism of distant organs.* Little damage is caused by the plugging of a vessel by an aseptic embolus, where collateral circulation may be readily established. More serious is the occlusion of end-arteries in the brain, lung, spleen, kidneys, etc., especially by septic emboli.

Endocarditis—physical signs: *Inspection* and *palpation* may reveal an increased or decreased *action of the heart.* Percussion later shows an increase of the *heart-dulness.* More valuable evidence is gained through *auscultation,* even early in the course of the disease. The *affection of valves* causes various anomalies of sound. The *mitral valve,* for which endocarditis shows a preference, is usually rendered insufficient, with the production of a *bruit* synchronous with the systole or first sound of the heart and heard with greatest intensity at the apex. When the heart is very weak the murmurs and also the first sound of the heart may be absent. The mitral valve may be affected in such a way as to cause stenosis, when the murmur would be presystolic. With hypertrophy of the heart the valve-sounds become accentuated.

Diagnosis: The symptoms usually of most value in diagnosis are chill, fever, pain in the region of the heart, palpitation, anxiety, headache, insomnia, and dyspnœa. Sometimes the semirecumbent posture assumed by the patient may excite suspicion of the presence of endocarditis. Upon physical examination the heart's action may be found increased or decreased, the apex-beat displaced, the heart-dulness increased, and there may be murmurs indicative of valvular disease. The history or knowledge of the existence of one of the infections, especially acute rheumatism, septicæmia, pneumonia, or tuberculosis, may aid in an individual case.

The **differentiation** between simple and septic endocarditis may sometimes be made by a bacteriological examination of the blood and urine. Evidences of stenosis of a valve would indicate chronic endocarditis, as does also affection of the valves at the base of the heart and of the right side of the heart, except when congenital. Evidence of tricuspid insufficiency would point to chronic endocarditis. Affection of the mitral valve, causing a murmur most intense at the

apex, usually soft blowing in character, and synchronous with the first sound of the heart, would be caused by acute endocarditis. Relative insufficiency is usually due to acute endocarditis.

Prognosis: The prognosis should be guarded, but is not necessarily fatal, even in the presence of grave complications. The outlook is bad in malignant cases. Sometimes benign cases, especially in early life, may entirely recover with the disappearance of even marked physical signs. More frequently acute cases become chronic. Cases of *simple endocarditis* usually recover. The prognosis is always worse in septic cases, especially when due to the streptococcus pyogenes. Complications, especially embolism, add gravity to the prognosis.

Endocarditis—treatment: Rest in bed must be absolute. The diet should be light—milk, malted milk, and the gruels. The sick-room should be well ventilated; the bed-clothing light but adequate. Unnecessary visitors should be excluded. Treatment should, if possible, be addressed to the underlying cause, usually one of the infections. In the presence of rheumatism or infection by the micrococcus pneumoniæ crouposæ, sodium salicylate may be administered, gr. v–x every two to four hours, best with whiskey or brandy. Palpitation that causes distress may be relieved by cold compresses over the heart. Phenacetin, gr. iij–v, may be given to quiet excessive action of the heart and relieve pain. More severe pain may be met with opium, best in the form of Dover's powder, which may be given to children in the form of the syrup. Exceptionally morphine is required. A flagging heart demands stimulation (see Heart-failure).

In septic cases, the *original depot* of infection should be destroyed, if possible. Curetting may be required for endometritis; deep urethral injections for posterior urethritis, prostatitis, cystitis; climate and tuberculin for tuberculosis; exsection for osteomyelitis.

SCLEROTIC ENDOCARDITIS (Chronic Endocarditis; Chronic Valvular Disease; Atheroma).

Etiology: Usually sclerotic endocarditis results from an acute endocarditis. An important *rôle* in etiology is played

by strain of the heart in overcoming obstruction offered to the circulation by arteriosclerosis, Bright's disease, etc. Muscular exertion may rupture a valve or one or more of the chordæ tendineæ through distention of the heart; but such an accident usually can occur only when the valves or chordæ tendineæ have been previously weakened by disease. Frequently a weak heart seems to be inherited.

Symptomatology: As in acute endocarditis, the inflammation may be confined to the wall of the heart and symptoms be altogether absent. Such cases are recognized first upon autopsy; or the nature of the disease may be suspected upon detachment of particles of a thrombus producing *embolism* in various parts of the body, especially the brain, retina, lungs, spleen, kidneys, organs in which there are endarteries. Rarely the valves may be affected without signs of the disease. Oftener the evidence of affection of the valves is overlooked because disease of the heart is not suspected.

The *general symptoms* of sclerotic endocarditis are due largely to the *condition of the heart-muscle.* There are failure in the general health, a lack of zest, early fatigue, palpitation, and rapid pulse and respiration upon slight exertion. The mitral valve is most frequently affected; there may be dyspnœa, and the individual "takes cold" easily, due to the disturbed nutrition. Sometimes there are cough, hæmoptysis, chilly sensations, sweats, loss of weight, anæmia—resembling very much the picture of tuberculosis. Frequently there is dyspepsia, which may be the chief complaint. Sometimes there is œdema, especially about the ankles, which may be the first symptom noticed by the patient. With the breaking of compensation the pulse becomes irregular and weak.

The **remote** or **secondary symptoms** of sclerotic endocarditis depend upon: (1) *Defective blood-supply*, evidenced by faintness, vertigo, syncope, early fatigue, increased secretion of uric acid. (2) *Stasis*, the dropsy first appearing about the ankles and extending over the body. There are increased frequency of the respiration, catarrh, dyspnœa, in bad cases cyanosis and hæmoptysis; hebetude, headache, later delirium and coma, and in extreme cases œdema of the brain. With *insufficiency of the tricuspid valve* there are dilatation and throbbing of the

jugular veins and other veins in the neck; the *liver* may show pulsation, which is more perceptible upon palpation. In the liver there is an increased formation of connective tissue; the organ at first becoming enlarged, and later the connective tissue contracting to constitute a true cirrhosis (cardiac cirrhosis). Extreme stasis causes the so-called "nutmeg liver." The *kidneys* become congested and enlarged, the urine diminished in quantity, sometimes amounting to anuria; in protracted cases the kidneys become cirrhotic. Affection of the *stomach* causes dyspepsia, anorexia, gastrectasia, etc.; affection of the intestines, chronic catarrh, constipation, hemorrhoids. (3) *Embolism* does not occur so frequently as in acute endocarditis.

Prognosis and treatment: See Valve-lesions.

VALVULAR DISEASE.

The **valves** are said to be *insufficient* or *incompetent* when they fail to close the opening so as to prevent a reflux of blood. In *stenosis* the valves offer obstruction to the normal flow of blood through the orifice.

In "relative" insufficiency the valves may remain normal; but the opening they guard is increased in size, through dilatation of the heart, so the valves are no longer able to prevent the return flow of blood.

Mitral Insufficiency.

Definition: A condition of the valves guarding the left auriculo-ventricular orifice, that permits some of the blood to return to the left auricle upon contraction of the left ventricle.

Etiology: The most frequent cause is endocarditis. Other causes are atheroma, myocarditis, fatty heart, and neoplasms.

Mitral insufficiency—symptomatology: A systolic *murmur*, synchronous with the first sound of the heart, is heard with greatest intensity at the apex. Sometimes this bruit is heard most distinctly toward the end of systole with the last forcible contraction of the left ventricle. Sometimes the sound seems to be continued into the second sound, constituting the *souffle paradoxal* (Paul), so named because of its resemblance

to a true diastolic murmur. A better term probably is *pre-diastolic* (Fraentzel). A long auricular appendage sometimes causes the murmur to be heard with equal intensity at the base of the heart, about two inches to the left of the region of the pulmonary valve-sound.

Regurgitation of blood through the mitral orifice into the left auricle, causes *hypertrophy of the left auricle*, which soon gives way to dilatation. *Dilatation* of the left auricle may cause the heart-dulness to extend upward as high as the second rib. The obstruction to the circulation increases the blood-pressure, not only in the left auricle, but also backward through the pulmonary veins and the pulmonary artery, to cause *accentuation of the pulmonary valve-sound.*

The *pulmonary valve-sound* is heard best to the left of the sternum, at the second costo-sternal junction or in the second interspace. Accentuation of the pulmonary valve-sound may also be caused by obstruction to the circulation in the lungs or by arterio-sclerosis. When hypertrophy of the right ventricle gives way to dilatation, accentuation of the pulmonary valve-sound may no longer be heard.

The extra work caused by the increased blood-pressure in the pulmonary artery causes *hypertrophy of the right ventricle.* The heart-dulness extends beyond the right border of the sternum. Hypertrophy of the right ventricle may compensate for a long time for the defect in the mitral valve. With hypertrophy of the right ventricle the heart comes to lie more upon its side and a larger surface rests upon the diaphragm, so that pulsation of the right ventricle may often be felt at the ensiform cartilage. The apex is dislocated to the left. Sometimes the right ventricle comes to form the apex of the heart.

Later, *compensation is broken*, the hypertrophy of the right ventricle giving way to *dilatation. The action of the heart becomes weaker, the pulse, cardiac impact, and apex-beat less perceptible.* Relative insufficiency of the tricuspid valve may give rise to *pulsation in the liver and the veins of the neck.*

Mitral Stenosis.

Definition: Obstruction of the left auriculo-ventricular orifice.

Etiology: Mitral stenosis is most frequently due to endocarditis; rarely to aneurism in the wall of the ventricle or of the valve; very rarely to neoplasms. Stenosis occurs often in early life and has been found at birth.

Symptomatology: *Obstruction* is offered to the circulation at the left auriculo-ventricular orifice. The symptoms bear a marked resemblance to those of mitral insufficiency. Insufficiency of the mitral valve is frequently present in mitral stenosis. *Stasis in the lungs gives rise to shortness of breath upon slight exertion*, such as climbing stairs. The individual *takes cold upon every exposure*. There are cough, expectoration of frothy mucus, and sometimes hæmoptysis. Lessened blood-supply to the brain is evidenced by *lack of concentration of the mind, apathy, and emotional disturbances*. At first the surface is pale, the mucous membranes anæmic; later there are venous stasis, cyanosis, icterus, and dropsy, first appearing about the feet.

The obstacle to the circulation offered by the diminution in size of the left auriculo-ventricular orifice causes increased blood-pressure within the left auricle, which is met at first by *hypertrophy of the left auricle*. This soon gives way to *dilatation*. *The blood-pressure is increased within the pulmonary vessels and lungs*, which early interferes with the aëration of the blood, causing *cyanosis* and predisposing the individual to *catarrh* of the respiratory apparatus and *hemorrhage*. The increased blood-pressure within the pulmonary artery causes distinct *accentuation of the pulmonary valve-sound*. *The second sound is frequently split*. *The right ventricle undergoes rapid hypertrophy*, and more readily becomes *dilated* than in mitral insufficiency. The *frémissement cataire* of Laennec, a peculiar presystolic or diastolic vibratory thrill, may be felt over the heart. As in mitral insufficiency, *dulness* may extend upward to the second rib and beyond the right border of the sternum. The *murmur*, which is heard in greatest intensity at the apex, is presystolic—that is, occurs just before the second sound of the heart. Hypertrophy of the right ventricle causes *accentuation of the tricuspid valve-sound*, heard best over the ensiform cartilage, which disappears upon dilatation of the right ventricle.

Dilatation of the pulmonary artery may result in *relative insufficiency of the pulmonary* (semilunar) *valves*, producing a diastolic murmur heard in greatest intensity over the third or fourth left interspace near the sternum. With dilatation of the right ventricle the tricuspid valves become incompetent, with consequent *venous stasis*, and pulsation of the veins in the neck. The liver becomes enlarged and may show pulsation.

On the part of the *kidneys*, the *urine* becomes diminished in quantity, high in color and specific gravity, and contains albumin. The action of the heart becomes weak, as is evidenced by the pulse, which becomes weak, irregular, and intermittent.

Aortic Insufficiency.

Definition: Imperfect closure of the aortic orifice, permitting regurgitation of blood from the aorta into the left ventricle.

Etiology: Insufficiency of the aortic valves is due to endocarditis or the process of arterio-sclerosis or atheroma. Dilatation of the aorta (aneurism) may give rise to relative insufficiency of the aortic valves.

Aortic insufficiency—symptomatology: Usually the *onset is insidious*. Frequently insufficiency of the aortic valves exists for a long time unrecognized. The first symptoms complained of by the patient may be nervousness, palpitation, pain, vertigo. Sometimes the onset is sudden, especially when the insufficiency is induced by severe strain, and is announced by precordial pain, intense dyspnœa, a diastolic murmur, and accentuation of the second aortic sound. The change from hypertrophy to dilatation is marked by the symptoms of heart-failure. Early symptoms are vertigo, headache, insomnia. There is pulsation of the carotids. There may be disease of the kidneys. Cerebral hemorrhage may be caused by arteriosclerosis or embolism. Hypertrophy of the right ventricle prevents for a long time symptoms on the part of the lungs. Later there may be attacks of dyspnœa, appearing first only upon exercise.

Inspection and *palpation* may reveal *enlargement of the*

heart, often to such a degree as to constitute the *cor bovinum*, or "ox heart," due to *hypertrophy of the left ventricle*. The cardiac impact is increased. The heart throbs.

Dulness may extend from the right border of the sternum to the left mammary line and from the second to the sixth rib.

With beginning failure of the left ventricle to perform its unusual labor, extra work is required of the *left auricle*, which soon gives way under the strain to cause increased blood-pressure in the pulmonary veins, lungs, pulmonary artery, and the right ventricle.

The *right ventricle* becomes hypertrophied and adds to the enlargement of the heart, which is caused chiefly by hypertrophy of the left ventricle.

Later the hypertrophy of the left ventricle may relieve the right ventricle of its extra work and cause the lung-symptoms to disappear. A *murmur*, synchronous with the second sound of the heart—therefore diastolic, sometimes pre-diastolic—is heard most distinctly upon the right of the sternum in the second or third intercostal space. The blood-pressure in the arteries is not long sustained by the aortic valves, and the pulse rises and falls abruptly with the beating of the left ventricle, constituting the *cannon-ball pulse*, first described by Corrigan, of Dublin, in 1830, and sometimes called Corrigan's pulse.

Aortic Stenosis.

Aortic stenosis is most frequently due to arterio-sclerosis or atheroma extending from the aorta to involve the aortic valves. The valves become thickened or calcified. Sometimes aortic stenosis is due to endocarditis, usually as an extension of the process which has first involved the mitral valves. Aortic stenosis is more common in age, but has been found at birth. Aortic stenosis rarely occurs alone—that is, without other diseases of the valves.

Aortic stenosis—symptomatology: Obstacle is offered to the exit of blood from the left ventricle, causing *increased blood-pressure within the left ventricle, with consequent hypertrophy.* The hypertrophy is not so great as in aortic insufficiency. The

apex-beat is displaced but little to the left, and appears in the sixth intercostal space. The action of the heart is stronger than normal. The impact of the heart is increased. With failing of compensation, the left ventricle becomes dilated, sometimes to such an extent as to cause relative insufficiency of the mitral valves. There follow hypertrophy and dilatation of the left auricle, increased blood-pressure in the pulmonary veins, lungs, and pulmonary arteries, and hypertrophy of the right ventricle, which may later give way to dilatation to cause insufficiency of the tricuspid valves.

A fremitus or thrill, the *frémitus cataire*, may be felt upon the right of the sternum over the second intercostal space, synchronous with the first sound of the heart, therefore systolic. A *murmur* is heard with greatest intensity over the second intercostal space to the right of the sternum, also synchronous with the first sound of the heart or systole. The second sound of the heart is feeble, in the absence of insufficiency of the valves. *The pulse is slow, small, and wiry.*

Tricuspid Insufficiency.

Insufficiency of the tricuspid valve is due to endocarditis or atheroma, and usually occurs in connection with affection of other valves. In the fœtus, the tricuspid valves are most frequently affected, probably because most work is thrown upon them. In extra-uterine life insufficiency of the tricuspid valves is due, as a rule, to relative insufficiency, caused by dilatation of the right ventricle.

Tricuspid insufficiency—symptomatology: The heart is enlarged, especially toward the right; there is distinct pulsation at the ensiform cartilage or in the epigastric region. Hypertrophy gives way to dilatation sooner than in mitral insufficiency. A *murmur* may be heard in greatest intensity at the ensiform cartilage. The second pulmonary sound is weak. Weakening of the accentuated pulmonary valve-sound in disease of the mitral valves indicates complicating insufficiency of the tricuspid valves. There is *venous pulsation*, especially in the jugular and subclavian veins, observable first at the root of the neck in the bulb of the jugular vein. The

venous pulse is presystolic-systolic. Upon overcoming the Eustachian valve pulsation appears in the liver and in the femoral veins. There may be cardiac cirrhosis of the liver and spleen. With weakening of the action of the right ventricle the blood-pressure becomes lowered. The supervention of *stasis* is marked by ectasia of the veins, cyanosis, and cyanotic induration of the internal organs—lungs, liver, etc. *Œdema* becomes manifest first about the ankles, and mounts up the extremities and trunk. There are periodic attacks of dyspnœa, *cardiac asthma*, due to defective circulation and consequent defective oxygenation of the blood.

Tricuspid Stenosis.

Stenosis of the tricuspid valve, or obstruction at the right auriculo-ventricular orifice, occurs most frequently in the fœtus. Occasionally cases have been reported in the adult, in the great majority of cases complicated with lesions of other valves, especially the tricuspid, mitral, and aortic. In the reported cases most of the patients have been of the female sex. Tricuspid stenosis in the fœtus is believed to be due to endocarditis, the result of rheumatism; a large proportion of the cases in the adult are due to rheumatism. Other cases are ascribed to the causes of disease of other valves.

Symptomatology: The symptoms of *tricuspid stenosis* are frequently overshadowed by the lesions of other valves and myocarditis. There is *extreme stasis* with marked dyspnœa, cardiac asthma, and cyanotic induration of the liver, spleen, and kidneys. Icterus may be more or less pronounced. A diastolic bruit may be heard in the region of the ensiform cartilage. The right ventricle does not become hypertrophied in pure tricuspid stenosis, but rather undergoes atrophy. Extreme distention of the right auricle causes an increase in the heart-dulness.

Pulmonary Insufficiency.

Definition: Incompetence of the pulmonary valves, permitting the regurgitation of blood from the pulmonary artery into the right ventricle.

Etiology: Insufficiency of the pulmonary valves is usually congenital. The condition may be acquired through endocarditis or arterio-sclerosis. Rarely cases may be due to trauma. A complicating pulmonary stenosis is the rule.

Pulmonary insufficiency—symptomatology: Dyspnœa and cyanosis soon become apparent. Hypertrophy of the right ventricle may for a time compensate for or overcome the insufficiency of the pulmonary valves. Soon the right ventricle begins to fail and symptoms develop on the part of the lungs. Catarrhal symptoms and pulmonary hemorrhage appear early. Tuberculosis, which occurs frequently in these cases, is to be ascribed rather to an accompanying pulmonary stenosis. The heart-dulness is increased, with *hypertrophy of the right ventricle.* The dulness may extend to or beyond the right border of the sternum. The apex-beat is dislocated to the left. Systolic pulsation may be readily recognized in the epigastrium and in the second intercostal space to the left of the sternum. A diastolic *bruit*, therefore, synchronous with the second sound of the heart, may be heard with greatest intensity in the second left intercostal space. Palpation may detect a diastolic fremitus or thrill. The pulse is regular.

Pulmonary Stenosis.

Stenosis of the pulmonary valves is the *most frequent congenital defect of the valves of the heart.* The condition is rarely acquired. Pulmonary stenosis is usually associated with other congenital anomalies, especially persistence of the foramen ovale and ductus arteriosus and perforation of the septum. Acquired cases occur usually in the course of endocarditis, after the affection of other valves.

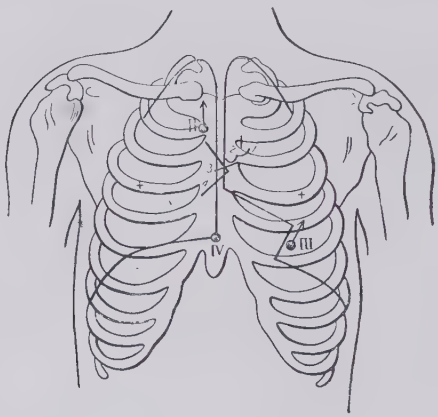
Pulmonary stenosis—symptomatology: *Hypertrophy of the right ventricle* causes dulness to become appreciable up to or beyond the right border of the sternum. There is epigastric pulsation. The apex-beat is slightly displaced. In the presence of perforation of the septum, some of the blood escapes to the left side of the heart, causing more or less hypertrophy of the left ventricle. Since the condition occurs in early life, there may be a distinct bulging of the chest. A

murmur, synchronous with the first sound of the heart, may be heard in the second left intercostal space. Congenital cases show cyanosis and usually arrested development of the body. Pulmonary stenosis predisposes the individual to tuberculosis, probably through imperfect development of the lungs more than through the defective blood-supply.

Combined Valve-lesions.

The following are the more frequent **combinations**: Insufficiency and stenosis of the mitral valve; insufficiency and stenosis of the aortic valve; stenosis of the mitral valve with insufficiency of the aortic valve; stenosis of the aortic valve with insufficiency of the mitral valves; insufficiency of both

FIG. 35.



1, 2, 3, 4, location of valves; I, II, III, IV, points where murmurs are heard in greatest intensity; arrows indicate direction in which murmurs are propagated.

mitral and aortic valves; stenosis of both mitral and aortic valves; tricuspid insufficiency with lesions of the mitral valve; tricuspid insufficiency with insufficiency of the aortic valves; tricuspid stenosis with other valve-lesions.

Diagnosis of valve-lesions: *Accidental murmurs* belong usually to anæmia or cachexia. During compensation there is no

evidence of stasis or a failing of the circulation, notwithstanding the lesion in the heart. The *breaking of compensation* is indicated by general degradation of health, loss of energy, inability to concentrate the mind, insomnia, headache, hebetude, sometimes vertigo, and symptoms on the part of the lungs. The pulse is increased in frequency and becomes weak. The surface of the body shows a lower temperature than normal. There is cyanosis; the veins become distended; there is enlargement of the liver and spleen.

In the *adult*, the diagnosis of valve-lesions has to do almost exclusively with the left side of the heart, the mitral and aortic valves. Tricuspid insufficiency is of especial importance, since it is usually secondary to affection of other valves, and, as a rule, indicates a failing heart.

Lesions of the right side of the heart, especially tricuspid stenosis and affections of the pulmonary valves, are congenital. Such lesions occur in early life, usually in association with other congenital defects.

Valve-lesions—Physical and other Signs.

Mitral insufficiency: (1) Systolic murmur, heard with greatest intensity at the apex. (2) Dilatation of left auricle, with dulness at the second rib to the left of the sternum. (3) Hypertrophy of the right ventricle, with dislocation of the apex-beat to the left, and dulness to the right of the sternum. (4) Accentuation of the second pulmonary valve sound.

Mitral stenosis: (1) Presystolic murmur at the apex. Usually cases of mitral stenosis show also mitral insufficiency; the murmur at the apex is then systolic and presystolic. (2) Dilatation of the left auricle, earlier and more complete than in mitral insufficiency, with dulness at the second rib to the left of the sternum. (3) Hypertrophy of the right ventricle, which gives way to dilatation sooner than in mitral insufficiency; dulness increased to the right of the sternum; dislocation of the apex-beat to the left and visible pulsation. (4) Accentuation and sometimes splitting of the pulmonary valve sound.

Aortic insufficiency: (1) Diastolic murmur in the second right

interspace. (2) Marked, often extreme, hypertrophy of the left ventricle. Visible carotid pulsation. (3) Cannon-ball pulse. (4) Alcoholism or syphilis, prominent factors in the production of arteriosclerosis and atheroma, upon which aortic insufficiency usually depends.

Aortic stenosis: (1) Systolic murmur in the second right interspace. (2) Hypertrophy of the left ventricle, with dislocation of the apex-beat downward and somewhat to the left. (3) Hard, wiry pulse. (4) Other diseases of the valves, especially aortic insufficiency, with which aortic stenosis is often associated.

Tricuspid insufficiency: (1) Systolic murmur in the region of the ensiform cartilage or epigastrium. (2) Dilatation of the right ventricle. (3) Venous pulsation, first observable upon effort, especially in the jugular bulbs and the liver. (4) Signs of a failing heart, cardiac asthma, cyanosis, edema; and symptoms on the part of the lungs, especially cough, shortness of breath, and hemorrhage. (5) Other diseases of the valves. Primary tricuspid insufficiency is sometimes encountered in the fœtus, but is very rare in adults.

Tricuspid stenosis: (1) Presystolic murmur at ensiform cartilage. (2) Extreme dilatation of the right auricle. (3) General venous stasis. (4) Congenital; rare.

Pulmonary insufficiency: (1) Diastolic murmur in the second left interspace. (2) Hypertrophy and dilatation of the right ventricle. (3) Congenital; rare.

Pulmonary stenosis: (1) Systolic murmur in the second left interspace. (2) Extreme hypertrophy and dilatation of the right ventricle. (3) Cyanosis—"blue births." (4) The most common congenital lesion of the heart.

Prognosis of valve-lesions: *Diastolic lesions* have a graver outlook than systolic lesions.

The prognosis is most favorable in mitral insufficiency, especially in childhood or when the lesion develops gradually. More grave is the outlook in aortic insufficiency, and still worse in aortic stenosis.

The prognosis in *mitral stenosis* is almost as bad as in lesions of the right side of the heart. The outlook is bad in *tricuspid insufficiency*, which usually indicates dilatation of the

heart (relative insufficiency). The outlook depends largely upon the response of the heart to cardiac stimulants, especially digitalis.

The prognosis is bad in *relative* aortic insufficiency, because the heart-muscle is weakened. Often there is arteriosclerosis.

Cyanosis and *dyspnœa* are bad signs, since they indicate a failing heart. A weak and irregular pulse is also ominous, when it may not be corrected by rest and heart-stimulants.

The *prognosis in embolism* takes color with the situation and character of the embolus. The outlook is bad in septic embolism. Organic valvular disease is made worse by pregnancy. The prognosis is aggravated by tuberculosis. The outlook depends largely upon the condition of the heart-muscle, the habits and age of the individual, and his ability to take proper care of himself. The outlook is bad in alcoholics. Congenital lesions of the valves of the right side of the heart give a bad prognosis.

Prophylaxis of valve-lesions: The power of the heart-muscle should be conserved as much as possible. The individual must avoid the two extremes of over-activity and inactivity. In acute cases or during exacerbations the patient should observe absolute rest in bed. With the establishment of a compensatory hypertrophy the heart must not be overtaxed. The individual should sleep more and work less, as a rule. The diet should be light and nutritious. In bad cases milk is the best diet. Sudden exertions and emotions and the use of heart-stimulants, including alcohol, tobacco, coffee, and tea, should be avoided.

Treatment of valve-lesions: Heart-stimulants should be withheld so long as there is perfect compensatory hypertrophy. If possible, the cause should be discovered and properly treated. Arteriosclerosis, rheumatism, tuberculosis, etc., should be properly treated. Absolute rest in bed should be enjoined before complete compensation is established, and also in cases of breaking compensation. In the presence of compensatory hypertrophy the individual should receive the instructions given under Prophylaxis of Valve-lesions, that the hypertrophy may be conserved. The patient should lead a

pleasant, quiet life. A change to a warm climate may be advisable, especially during the winter and fall months.

Some cases show marked improvement following the regular use of the *warm bath*. Constipation should be avoided; the bowels should move once or twice a day. Much may be gained by carefully graded exercise, best in the open air, never carried to the point of exhaustion or strain. The heart-muscle is thus toned, just as any other muscle may be strengthened by exercise. Palpitation may be relieved by the application of cold, most conveniently in the form of the ice-bag or Leiter coil. Sometimes the patient may find it advisable to carry a hollow tin shield or flask filled with cold water in an inside pocket over the heart. Tumultuous action of the heart may sometimes be controlled by the bromides, especially sodium bromide, gr. xx–xl. Pain varies greatly in intensity in different cases. Slight pain may be relieved by a mustard-plaster, or in more stubborn cases by a belladonna-plaster, or the administration of phenacetin, the salicylates, or salol. Severe pain may be relieved by morphine or the nitrates. Amyl nitrite, gtt. ij–v, may be inhaled from a napkin or handkerchief. Repeated attacks are prevented by the use of nitroglycerin, gtt. j–v of a 1 per cent. solution three or four times daily. A failing heart demands stimulation: strychnine, caffeine, camphor, digitalis, or strophanthus. Dropsy that does not disappear upon rest may be relieved by caffeine, digitalis, or sodium salicylate, theobromine (diuretin), and the use of calomel. Extreme dropsy is relieved by puncture, probably best by one or two canulæ in each lower extremity.

Dyspnœa usually disappears under stimulation of the heart: camphor, valerian, or Hoffmann's anodyne. Pain and dyspnœa are both relieved at once by morphine, best given hypodermatically.

NEUROSES OF THE HEART.

A **neurosis** is a disturbance of function in the absence of any *demonstrable organic* lesion. The action of the heart is regulated to meet the requirements of the body by motor, vasomotor, and sensory impressions received through branches

from the vagus, superior and inferior laryngeal nerves; the cervical and first dorsal ganglia; the pulmonary plexus, and occasionally from the descending part of the hypoglossus (Luschka). The sensory ganglia of the heart belong to the sympathetic system.

ARRHYTHMIA.

Arrhythmia, or irregular action of the heart, may vary from the occasional loss of a beat to the great irregularity known as *delirium cordis*. The *pulsus bigeminus* is marked by the occurrence of two beats followed by an intermission; the *pulsus trigeminus*, by three beats followed by an intermission. The *pulsus alterans* consists of regularly alternating strong and weak beats. Various combinations exist. A *pulsus bigeminus alterans* would consist of a strong beat and a weak beat, followed by an intermission. The *tremor cordis* is marked by very rapid pulsation, giving one the impression of a vibration or tremor. Normally, the pulse is stronger upon inspiration; but it may be weaker, constituting the *pulsus paradoxus*. A regular arrhythmia is known as *allorrhythmia*.

Etiology: Arrhythmia may be caused directly by *lesions in the brain and spinal cord* affecting the centres of the pneumogastric and accelerator nerves. Among such lesions are meningitis, apoplexy, tumors, and abscess. *The nerves may be pressed upon* by enlarged glands in the neck, including the thyroid and thymus glands, or by tumors or aneurism. Aneurism of the arch of the aorta may produce arrhythmia. Arrhythmia may also occur in *pericarditis*, *myocarditis*, *endocarditis*, *arterio-sclerosis*, *anæmia*, *chlorosis*, *leukocythæmia*, or in cases of *distention of the stomach or intestines with gas*, or *upward displacement of the diaphragm by fluid*. *Just before death* the heart may become irregular as well as weak. The most common *reflex causes* are injuries of the abdominal organs, kidneys, or uterus, and severe pain. Among the *toxic causes* are the infectious diseases, especially typhus fever, scarlet fever, cerebro-spinal meningitis, diphtheria; Bright's disease and rheumatism; the abuse of alcohol, tobacco, coffee,

and tea. Arrhythmia may be caused by *emotions*, especially home-sickness, love-sickness, and domestic infelicities.

In **diagnosis** the most important item is the discovery of the cause, the recognition of which takes the arrhythmia out of the category of diseases of obscure or unknown causes and makes it a symptom.

The **prognosis** and **treatment** depend upon the cause.

PALPITATION.

Palpitation is a beating of the heart that is felt by the patient.

Etiology: The *direct causes* are *organic diseases of the heart*, especially those due to arterio-sclerosis or Bright's disease; *irritations in the brain and spinal cord*, including the emotions and organic diseases of the central nervous system, especially tabes dorsalis, disseminated sclerosis, progressive paralysis, epilepsy, Basedow's disease, *neurasthenia and hysteria; irritation of the nerves supplying the heart*, caused most frequently in the neck by tuberculous glands. The *reflex causes* are located chiefly in the abdomen and pelvis. Thus palpitation may be caused by affection of the stomach, distention of the stomach or intestines with gas, constipation, prostatitis, salpingitis, sexual excesses and perversions, puberty, and severe pain. The more common *toxic causes* are the abuse of alcohol, tobacco, coffee and tea, Bright's disease, gout, anæmia, leukocythæmia, chlorosis, scurvy, and plethora.

The **diagnosis** concerns chiefly the discovery of the cause, upon which the *prognosis* and *treatment* largely depend.

Treatment: During the attack the individual should observe absolute rest, best in bed. The cause of emotions, especially anxiety, should be removed if possible. Excessive action of the heart may be quieted by cold applications, best in the form of the ice-bag or cold compresses, placed over the heart. Mild cases are relieved by the use of the sodium benzoate of caffeine, gr. iij–v, or sodium bromide, gr. xx–xl largely diluted, or valerian or ergotin. In bad cases the attack may be cut short by the administration of brandy, Hoffmann's anodyne, the compound spirit of ether, or the

aromatic spirit of ammonia, or by morphine subcutaneously. In all cases the cause of the palpitation should be sought and removed or properly treated.

TACHYCARDIA.

Tachycardia is an increased rapidity of the action of the heart to more than 100, sometimes reaching 200 or even 300 beats per minute. Increased frequency of the beating of the heart may be due to Basedow's disease, some change in the nerve-centres, or continuous toxic irritation, and occurs sometimes in the absence of demonstrable disease. Tachycardia may be periodic or transitory in cases of fever, especially the infections, in convalescence and in degradation of the blood, anæmia, etc. Attacks of paroxysmal tachycardia may be due to the accumulation of toxins, the symptoms disappearing upon the elimination of the toxins.

Tachycardia—etiology: Among the direct causes, acting chiefly upon the pneumogastric nerve, are the *emotions*, which interfere with the inhibitory action of the cerebrum; *organic affections of the brain*, meningitis, sclerosis, tumors, softening; *tumors in the neck*, enlarged glands; and *inflammations and degeneration of the heart-muscle*. Rarely tachycardia may occur *reflexly* from disease in other organs. The more common *toxic causes* are Bright's disease, tuberculosis, rheumatism, and occasionally gout and lead-poisoning. Sometimes the cause of tachycardia may not be found. Such cases are ascribed to toxins of cryptogenetic origin, or to molecular changes in the nerve-centres similar to those which are supposed to exist in epilepsy and migraine.

The **prognosis** as to life is good, so far as the tachycardia is concerned; but death may be due to the cause of the tachycardia. Cases usually prove obstinate to treatment.

Treatment: The attack may be relieved in some cases by compression of the vagus in the neck. Sometimes attacks may be aborted by the use of morphine and atropine, nitroglycerin, or inhalations of amyl nitrite. In addition to the treatment of the attack, effort should be made to discover and remove the cause.

BRADYCARDIA.

Bradycardia is a reduction of the pulsation of the heart to 40 or less beats per minute.

Etiology: *Permanent bradycardia* may be caused by *irritation of the pneumogastric nerve*, which may occur in pachymeningitis, cerebro-spinal meningitis, trauma, hydrocephalus, tumors, and abscesses. Bradycardia and epilepsy often coexist, when both are usually due to the same cause. *Temporary bradycardia* occurs in the infections, especially typhoid fever, diphtheria, pneumonia, erysipelas, and rheumatism; in puerperal fever, jaundice; and in cases of absorption of toxins, as from the intestinal canal in cases of occlusion. Paroxysmal tachycardia is usually attributed to nervous causes. Among the *direct causes* of bradycardia are all those *conditions, including disease or injury of the brain, which may cause irritation of the vagus nerve.* *Reflex* bradycardia arises chiefly from some abdominal irritation. *Toxic* cases are due to uræmia, icterus, and the toxins. *Chronic* cases are frequently due to lead-poisoning or gout.

The **prognosis** should be guarded, and depends largely upon the cause.

Treatment: If possible, the cause should be found and removed or properly treated. Heart-stimulants are indicated only in the presence of a failing heart.

ANGINA PECTORIS.

Angina pectoris is marked by excruciating pain in the region of the heart, radiating to the left shoulder and arm.

Etiology: The pain of angina pectoris is the cry of the tissues for fresh blood. The blood-supply of the heart is partially or completely cut off by sclerosis involving the coronary arteries or by an atheromatous plate in the aorta at the origin of the coronary arteries.

Symptomatology: The disease develops slowly, as an arteriosclerosis or atheroma, but the attack comes on suddenly and with great severity. There are no premonitory symptoms. *The individual is literally transfixed with pain in the region of*

the heart and radiating to the left shoulder and arm. There is extreme anxiety. The attack may last from a fraction of a minute to several hours; as a rule, but a few minutes, and usually terminates abruptly.

Diagnosis: The extreme pain in the region of the heart and the anxiety during the attack are characteristic. There may be found some evidence of arterio-sclerosis or disease of the heart, myocarditis or valvular disease, especially stenosis of the aortic valves. The patient is usually past middle life or is an individual in whom the changes of age have been precipitated.

The **prognosis** is grave; but even bad cases may recover.

Angina pectoris—treatment: During the attack the individual instinctively observes absolute rest. Further relief of the arterial tension may be secured by the use of amyl nitrite by inhalation, gtt. ij–v, which should be discontinued with the cessation of the attack. Suitable doses of the amyl nitrite may be carried by the patient in the form of the so-called "pearls," which are broken and inhaled from a handkerchief. Persistent attacks may call for the use of the anæsthetics, preferably ether. Nitroglycerin may be given to prevent attacks, but acts too slowly for satisfactory use during attacks. Sodium nitrite and potassium nitrite are sometimes used. Protracted cases may demand the use of morphine.

PSEUDO-ANGINA.

Pseudo-angina usually occurs before the meridian of life, most frequently in subjects of hysteria or neurasthenia.

Etiology: Among the direct causes are affection of the branches of the pneumogastric nerve supplying the heart, and the cerebral changes which find expression in hysteria and neurasthenia. Emotional excitement plays a prominent rôle. Some cases may be traced to a reflex or toxic cause. The resemblance of the location of the pain in some cases to the distribution of herpes zoster has led Mackenzie to locate the cause in an infection affecting the posterior roots of the spinal nerves, especially the ganglia. The reflex causes are found chiefly in the alimentary canal, marked by associated dys-

pepsia or indigestion; frequently in the uterus and ovaries, with dysmenorrhœa, salpingitis, etc. Among the toxic causes are alcohol, tobacco, coffee, tea, gout, and lead-poisoning.

Symptomatology: Pseudo angina differs from true angina chiefly in the *predominance of emotional symptoms* and symptoms on the part of the vaso-motor system—vaso-motor angina.

Diagnosis: The emotions, the age and sex of the patient, and the existence of other evidence of hysteria or neurasthenia suffice to make the diagnosis.

Prognosis: Good.

Treatment: The treatment should be directed especially to the underlying cause. The attacks may be relieved by the administration of valerian, the bromides, Hoffmann's anodyne, sometimes by hot or cold applications or the use of a mustard-plaster.

DISEASES OF THE BLOODVESSELS.

ACUTE ARTERITIS.

The arteries may suffer acute inflammation, the process beginning in the vaso-vasorum. Such inflammation is seen most frequently in the acute infections, especially typhoid fever, pneumonia, and influenza; less frequently in smallpox, scarlet fever, and diphtheria. The inflammation may cause occlusion of the vessel, which may be followed by necrosis or gangrene; or the inflamed artery may become a depot of infection, giving rise to disseminated metastases and septicæmia.

ACUTE AORTITIS.

Inflammation of the wall of the aorta is accompanied by dyspnœa, marked by long, painful inspiration and short expiration. The discomfort varies in intensity from a feeling of constriction in the throat to intense pain, that may even resemble the agony of angina pectoris. The ascending aorta becomes dilated and elongated, so that the heart is displaced to the left, and the arch of the aorta sometimes becomes prominent in the neck, and the subclavian arteries may be detected by palpation above the clavicles.

The **treatment** is rest, with the relief of pain by opium when necessary, and the relief of the dyspnœa by the inhalation of amyl nitrite or ethyl iodide.

ARTERIO-SCLEROSIS.

Arterio-sclerosis is a degeneration of the walls of the arteries, frequently associated with atheromatous, calcareous, or fatty changes. The structural elements of the vessel-wall are substituted by connective tissue.

Etiology: The more important causes of arterio-sclerosis are alcohol, syphilis, rheumatism, and gout. Most cases are found in the aged. The disease is more common in the two extremes of society—the rich and the poor. Both hard work and high living are prominent causes. Arterio-sclerosis also occurs frequently in lead-poisoning, diabetes, and after the infections, especially typhoid fever, malaria, influenza, scarlet fever, and diphtheria. Other causes of arterio-sclerosis are bad habits and depressing emotions, especially worry.

Symptomatology: Arterio-sclerosis may exist even in marked degree and show no symptoms. One of the most prominent early symptoms is *increase in the blood-pressure*, which may be recognized at the pulse by the finger; or in finer degrees and more accurately by means of the sphygmograph and arteriometer. Sometimes *the vessels may be felt as hard cords*, resembling slate-pencils.

In *general arterio-sclerosis*, as in *arterio-capillary fibrosis*, the *blood-pressure is increased* and the *heart becomes hypertrophied*. The cardiac impulse is greater, the apex of the heart displaced; the *aortic valve sound*, which is the second sound at the base of the heart to the right of the sternum, *is accentuated*, and the *diameters of the heart are increased, especially to the left*. Later the hypertrophy gives way to dilatation, with a loss of all the signs except the evidence of an *increased size of the heart*. Elongation of the aorta may permit a *change of position of the apex-beat*. Thus, an apex-beat which is found in the normal position with the patient in the upright posture may be changed as far to the left as the axillary line when the individual lies upon the left side.

Atheroma of the aorta may diminish the supply of blood to the body. There may be present *aneurism of the aorta. Atheroma at the orifice of the coronary arteries* may give rise to arrhythmia, heart-failure, or angina pectoris.

Atheroma of the aorta is frequently associated with atheromatous changes in the aortic valves.

Sclerosis of the *coronary arteries* causes palpitation, especially after meals, and dyspnœa upon even light exercise. In some cases physical effort is followed by pain under the sternum, radiating to the left arm.

When the process involves the *brachial and intercostal arteries*, the blood-supply to the *respiratory muscles* is lessened, and there result ossification of the costal cartilages and pulmonary emphysema.

Sclerosis of the *pulmonary artery* occurs especially in cases of chronic stasis of the lung, as in mitral stenosis and tuberculosis. There are paroxysmal dyspnœa, palpitation, vertigo, and cyanosis, with hypertrophy of the heart, especially the right ventricle.

Arterio-sclerosis in the *kidneys* causes a granular atrophy, which begins as a degeneration in the small arteries.

Arterio-sclerosis is manifested on the part of the *brain* by alteration of disposition and mental disturbances. The individual becomes irritable and dejected. Mental strain causes exhaustion sooner than formerly. The mental faculties become weakened; there are impairment of motion, vertigo, insomnia, sometimes transitory aphasia, delirium, delusions; and at last the evidences of softening of the brain. Hemiplegia and apoplexy may occur at any time. Affection of the small vessels is usually present in chronic myelitis, tabes, multiple scleroses, syringomyelia, progressive paresis, and senile dementia.

Arterio-sclerosis of the *vessels of the extremities* may cause a lowering of the surface-temperature of the part supplied by the affected vessels, with cyanosis and gangrene. The hard arteries may be palpable.

Diagnosis: The most important symptoms in diagnosis are the evidences of hypertrophy of the right ventricle and increased blood-pressure, in the absence of any other discoverable cause, such as valvular disease, Bright's disease, etc.

Affection of the superficial arteries may be detected by palpation. There may be the evidence or history of some etiological factor, such as syphilis, alcoholism, hard work, saturnism, gout, etc. Usually the earliest sign is increased blood-pressure. Later there is dyspnœa upon slight effort, palpitation, precordial anxiety, coldness of the extremities, sensations of numbness, crises of pallor, and violent headache. There is early accentuation of the aortic valve sound, hypertrophy of the heart, and later dilatation.

Atheroma of the coronary arteries causes deficient nutrition of the body, with consequent marasmus, through a failing of the action of the heart. On the part of the heart there may be angina pectoris.

Chronic aortitis shows dyspnœa upon slight effort, which may not be accounted for by disease of the heart. In addition there may be the symptoms of arterio-sclerosis in other parts of the body, or the evidence or history of some cause of arterio-sclerosis.

The arcus senilis is an expression of arterio-sclerosis.

The Röntgen ray may reveal the deposits of arterio-sclerosis.

The **prognosis** is grave. Much depends upon the location and degree of the sclerotic changes, and the ability of the individual to regulate his life.

Prophylaxis demands moderation in eating, drinking, and exercise, the avoidance of bad habits, and the proper treatment of obesity, diabetes, lead-poisoning, and gout.

Arterio-sclerosis—treatment: The individual should live a life temperate in all things. If possible, the cause of the arterio-sclerosis must be discovered and removed. Of drugs, the iodides are the most valuable. Potassium iodide or sodium iodide, which is less depressing, or probably better the tincture of iodine, gtt. x in a wineglassful of sweetened water, may be given before meals. Systematic, carefully graded exercise, best out of doors, is of considerable value. Further treatment is symptomatic.

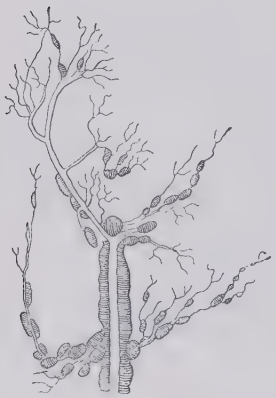
ANEURISM.

Aneurism, a circumscribed dilatation of an artery, occurs most frequently in the *external arteries*. In order of fre-

quency, aneurism occurs in the popliteal, crural, carotid, and the axillary arteries.

Internal aneurism occurs, in the order of decreasing frequency, in the aorta, subclavian, innominate, and pulmonary arteries. The *cerebral* arteries are affected as follows, in the order of frequency: the middle cerebral, basilar, internal carotid, anterior cervical, posterior communicating and anterior communicating, the vertebral, posterior vertebral, and the inferior cerebellar.

FIG. 36.



Dissecting aneurism on a cerebral vessel, simulating miliary aneurisms (Schmaus).

Dissecting aneurisms are due to rupture of the internal and middle coats of the artery, the blood making a channel between the middle and outer coats, or between the laminæ of the middle coat.

Miliary aneurisms, usually consisting of a large number of small dilatations, are found especially in the brain.

Etiology: The chief causes of aneurism are syphilis, alcohol, gout, rheumatism, strain, and depressing mental emotions.

Symptomatology: The disease may remain latent for a long time. Twenty per cent. of cases show no symptoms.

The chief symptoms of *aneurism of the aorta* are *pain, paræsthesiæ, paresis, dyspnœa, cyanosis, hoarseness, dysphagia, palpitation*, the presence of a *pulsating tumor*, and *pressure-symptoms.*

The tumor may cause various symptoms by pressure upon the œsophagus, branches of the vagus nerve, the intercostal nerves, branches of the brachial plexus, the spinal cord (after erosion of the vertebræ), the intestines, liver, bile-ducts, kidneys, and ureters. The *apex-beat* is displaced to the left and downward when the patient lies upon the left side. The left radial and carotid *pulse* is feeble or retarded.

Physical examination reveals a *thrill or bruit.* Rarely the

bruit may be heard only upon placing the bell of the stethoscope in the patient's mouth.

In aneurism of the *abdominal aorta* the pain is usually more diffuse. The chief pressure-symptoms are stenosis or obstruction of the intestine, biliary or renal colic, ascites, *pain* (gastralgia, enteralgia), paræsthesiæ, paresis, and paralysis. The *pulse* is retarded in the *crural arteries*. A pulsating *tumor* is found in the abdomen; but not every pulsating tumor in the abdomen is an aneurism.

Aneurism of the *pulmonary artery* usually occurs in connection with arterio-sclerosis and mitral stenosis.

Rupture of an aneurism is followed by immediate collapse.

The **diagnosis** depends upon the pressure-symptoms, pain, palpitation and dyspnœa, dislocation of the apex-beat, the detection of a pulsating tumor, and the characteristic thrill or bruit. Aneurism differs from tumors in causing more rapid erosion of bone than of the soft structures. The discovery of an etiological factor is often a valuable point. Doubtful cases may call for aspiration with a fine needle.

The **prognosis** is grave, but not necessarily fatal. The aneurism may lead to a fatal termination through rupture; or the sac may become obliterated and the individual recover.

Treatment: The blood-pressure should be lowered by absolute rest and a light diet. Medicinal treatment consists largely in the use of iodine, in the form of the tincture or the potassium or sodium salt. Further treatment is symptomatic or surgical. Quiescent cases should be let alone; cases that are progressing unfavorably may be relieved by surgery.

THROMBOSIS.

Definition: More or less complete occlusion of a vessel by a blood-clot. The process may occur in any part of the vascular system, most frequently in the arteries and veins.

Etiology: Thrombosis depends upon stasis of the blood and injury to the vessel-wall or blood by toxins or micro-organisms. The process occurs most frequently in the infections—septicæmia, tuberculosis, carcinoma, and Bright's disease.

The **symptoms** depend upon the location and character of the thrombus.

The **diagnosis** is made by the existence of an infection and the evidence of the cutting off of the blood-supply to a part of the body, and, in the case of septic embolism, by the occurrence of a depot of infection at the point of embolism with the usual symptoms of sepsis.

The **prognosis** depends upon the character and cause of the thrombus, its location, and the general condition of the patient.

Treatment demands rest and address to the cause. Otherwise the treatment is symptomatic.

EMBOLISM.

Definition: Interference with the circulation, caused by the arrest of an *embolus* or particle of foreign material in the circulation.

The **symptoms** of embolism vary with the character of the embolus and the location of the embolism. The embolus may be composed of fat, air, particles of a thrombus, etc. The chief sites of embolism are the brain, lungs, coronary arteries, splenic artery, mesenteric arteries, the kidneys, tympanum, and retina. There is interference, more or less marked, with the function of the organ affected.

Embolism of the brain occurs most frequently in the branches of the left carotid. Embolism of a large branch causes apoplexy, abolition of consciousness, coma, and hemiplegia. Septic emboli cause abscess, usually with meningitis.

Embolism of the lungs is announced by severe pain, sympathetic vomiting, and dyspnœa; sometimes with cyanosis, convulsions, and syncope (see Infarction of the Lung).

Embolism of the liver: Chill, fever, and icterus, with pain, swelling, and tenderness in the region of the liver.

Embolism of the spleen: Chill, fever, and severe pain in the spleen, which is enlarged and tender.

Embolism of the kidneys: Chill, fever, vomiting, and pain in the kidneys, with albuminuria and hæmaturia.

Embolism of the mesenteric arteries: Abdominal pain, diarrhœa, and the appearance of blood in the fæces.

Embolism of the retina: Sudden blindness. An infarction may be recognized upon ophthalmoscopic examination. A septic embolus may cause panophthalmitis with destruction of the entire globe. In such cases the affection of one eye is usually followed by affection of the other eye.

Embolism of the skin: Eruptions of various sorts appear—roseola, urticaria, petechia, ecchymosis—sometimes resembling the eruption of measles, scarlet fever, variola (pustules), or pemphigus (bullæ). In some cases there are sweating and sometimes desquamation. The skin of the lower extremities is most frequently affected, although the skin over the entire body may suffer from embolism.

Differential diagnosis: Embolism differs from thrombosis, as a rule, in being of more sudden onset. Embolism occurs especially in heart-disease, thrombosis in arterio-sclerosis. Embolism is further distinguished by affection of various organs. Embolism of the brain is differentiated from cerebral hemorrhage by the more sudden onset without prodromata, preference for early life and the female sex, and the connection with valvular disease.

The **treatment** of embolism demands rest and address to the cause. Further treatment is symptomatic.

PHLEBITIS.

Definition: Inflammation of the veins.

The **symptoms** vary with the vein affected and the character of the inflammation. *Femoral phlebitis* gives the symptoms of a thrombosis—arrest of circulation, and œdema of the leg, constituting the condition known as *phlegmasia alba dolens*. Such a phlebitis occurs most frequently in the puerperium. *Umbilical phlebitis* usually causes pain, fever, anorexia, hemorrhage, and collapse. Severe cases may show icterus. *Pyophlebitis* is a purulent inflammation caused by infection of a vein (*e. g.*, the portal), marked by the usual symptoms of septicæmia: chills, sweats, rapid weak pulse, clouded sensorium, diarrhœa, and collapse. There may be pain, icterus, ascites, dilatation of superficial veins, hemorrhoids, and hemorrhage from the bowels. Abscess may be differentiated by aspiration.

The **treatment**, further than rest and address to the cause, is symptomatic.

VARICES.

Varices, or phlebectasiæ, are more or less circumscribed dilatations of the veins, somewhat analogous to aneurisms in arteries. The veins become elongated and tortuous. Varices occur most frequently in the lower extremities, pelvis (broad ligaments), spermatic cord, prostate gland, and lower part of the rectum (hemorrhoids). A familiar example is the caput Medusæ, caused by cirrhosis of the liver and pyophlebitis.

Etiology: The chief causes are gravity, constriction of veins, age, syphilis, strain, alcohol, tobacco, lead-poisoning, and gout.

The **symptomatology** varies with the location and extent of the affection. There may be paræsthesia, neuralgia, alterations of the secretions, proctitis, venous stasis, elephantiasis, and various kinds of eruptions and ulcers.

Treatment calls for rest and gentle compression. An affected member should be elevated. Compression may be secured, in affection of the lower extremity, by the elastic stocking, and by the use of the suspensory bandage in affection of the spermatic cord. Further treatment is surgical.

DISEASES OF THE MEDIASTINUM.

The more important **diseases of the mediastinum** are mediastinitis or inflammation of the mediastinum; abscess of the mediastinum and mediastinal neoplasms. The principal neoplasms found in the mediastinum are sarcomata, carcinomata, dermoid and hydatid cysts. The mediastinum may also be the seat of syphilitic or tubercular growths.

CHAPTER V.

DISEASES OF THE BLOOD.

In this division will be considered the diseases characterized by changes in the blood or which are believed to be due to diseases of the blood; or of organs having some intimate connection with the blood.

The *parasitic diseases* of the blood have received consideration under Distomiasis, Filariasis, Malaria, etc.

The *normal number* of *red blood-corpuscles* in the adult is 4,000,000–5,000,000; in the infant, 6,000,000–8,000,000 per cubic millimetre. The number of corpuscles increases with the altitude. Thus, Laache found at Christiana, at the sea-level, 4,970,000 corpuscles per cubic millimetre; Viault, at Cordilleras, 4392 feet above sea-level, found 8,000,000.

The *percentages* of the *various kinds of leukocytes*, in adults and infants, according to the investigation of Shattuck and Cabot, are as follows:

	Adults.		Infants.	
Polymorphonuclear neutrophiles	60–70	per cent.	25–40	per cent.
Lymphocytes	18–30	"	40–60	"
Large mononuclear and transitional	4–8	"	6–12	"
Eosinophiles	0.5–4	"	1–10	"

PLETHORA.

The **red blood-corpuscles** show a *comparative increase* when the blood becomes more concentrated through the loss of its watery elements. Such a comparative increase of the corpuscles is seen in severe cases of diarrhœa, cholera, and in cases of excessive vomiting, sweating, etc. A similar comparative increase of the cellular elements of the blood may be produced by exercise, massage, or electricity, which cause increased blood-pressure and force the serum out of the vessels.

An *absolute increase* of the red blood-corpuscles is found in

the new-born, or may be caused by residence in high altitudes, delayed menstruation, cyanosis, myxœdema, and phosphorus-poisoning. The number of corpuscles per cubic millimetre may be increased by increased blood-pressure. True plethora should not be confounded with the "plethoric habit" due to vaso-motor disturbance or venous stasis.

ANÆMIA.

Anæmia may be roughly divided into *primary* anæmia, in which the chief etiological factor is believed to be in the blood or some organ in intimate connection with the blood; and *secondary* anæmia, in which the cause is probably not in the blood or in the so-called blood-making organs.

Primary anæmia includes chlorosis and pernicious anæmia.

Secondary anæmia includes the anæmias caused by hemorrhage, poisoning, the infectious diseases, malignant diseases, and the anæmias found in the insane.

CHLOROSIS.

Definition: An anæmia sometimes marked by a yellowish-green tint, occurring most frequently in girls about the time of puberty, seventeen to twenty-three years of age.

Etiology: The chief factors in causation are the infectious diseases, especially tuberculosis; constipation and the absorption of ptomaines, and bad hygiene. Many cases are ascribed to "cold," change of climate, and emotional disturbance or bad habits. The disease is believed to bear a relation to the establishment of ovulation. Often heredity seems to play a rôle.

Chlorosis—symptoms: The patient comes to feel *tired and sleepy all the time and loses interest in life;* there are *headache, palpitation, anorexia*, sometimes nausea and vomiting, frequently *perverted appetite, constipation*, and dysmenorrhœa or amenorrhœa. The tongue is coated, the breath foul. The *skin and mucous membranes are pale, with a yellowish, greenish, or bluish tint.* The *blood* is paler and more fluid than normal; there may be a slight diminution in the number of the red

blood-cells; usually the number of white blood-cells remains about normal; the number of blood-plates is increased. The most marked change in the blood is the *great reduction in hæmoglobin*. Usually there is a *soft systolic murmur*, heard most distinctly over the pulmonary valves. The second pulmonary sound is accentuated. The bruit de diable may be heard over the bulb of the jugular vein. There is *increased frequency of respiration*, even in the absence of exercise, as in rest and sleep. Thrombosis may occur, most frequently in the femoral vein. The plugging of a cerebral sinus may cause death.

The **diagnosis** is easy in the presence of typical changes in the blood. Before such changes occur the diagnosis is difficult. In all cases an attempt should be made to rule out pernicious anæmia and the secondary anæmias.

The **prognosis** is usually favorable.

Chlorosis—treatment: Bad cases should be confined to bed. Persistent cases may require a change of climate. Usually the disease responds readily to iron. A good form of iron is the improved Blaud pill. Arsenic may also be given, probably best as Fowler's solution or Roncegno water. Constipation is relieved by cascara or the pill of aloes and iron. Symptoms on the part of the stomach may be met with lavage and the administration of dilute hydrochloric acid, gtt. x-xx in a wineglassful of water before meals. Sometimes it is advantageous to use some bitters, the infusion of condurango, calumbo, absinthe, or the aromatic tincture of rhubarb. Bad habits and bad hygiene should be corrected. Nux vomica or gentian with bicarbonate of sodium often give good results.

PERNICIOUS ANÆMIA.

The **etiology** is obscure. Upon the discovery of a *cause*, most observers no longer classify cases as pernicious anæmia. Thus, many cases, which formerly would have been classified as pernicious anæmia, are now known to be infections by animal or vegetable parasites—*e. g.*, helminthiasis, malaria, tuberculosis, syphilis, etc.

Pernicious anæmia is most frequent in middle life, thirty

to sixty years, although cases have been reported in infancy and old age.

Pernicious anæmia—symptomatology: The disease comes on gradually with *pallor*, sometimes with a light icterus. The patient complains of *weakness and dyspnœa*. *Œdema* appears first about the ankles and eyelids; *later there is effusion into the serous cavities*. Notwithstanding the anæmia and degradation in strength, the *nutrition of the body suffers no or only slight impairment*. There may even be some *fat* apparent. Emaciation is the exception. As a rule, there is more or less irregular continuous fever. There is usually anorexia and an offensive breath, and frequently there are periods of nausea and vomiting. Occasionally patients complain of a voracious appetite. There may be constipation or diarrhœa. Physical examination may show enlargement of the liver and spleen. The most frequent symptom on the part of the nervous system is *headache;* there may be neuralgias, paræsthesiæ, pareses, apoplectiform attacks and delirium, numbness, tingling, lancinating pains, spasms, and delusions and hallucinations of sight and hearing. *Hemorrhage* occurs often, especially from the nose and gums. Retinal hemorrhage is not infrequent. *Palpitation, vertigo, and faintness* are common. Systolic *anæmic murmurs* may be heard over the base of the heart. Some cases show the Corrigan pulse. Examination of the chest may reveal the moist râles of passive congestion, and sometimes the presence of hydrothorax.

Most important are the symptoms on the part of the **blood**. The blood is *pale and thin*. The number of red blood-corpuscles varies from 1,000,000 to 500,000 or less per cubic millimetre. *The reduction of red blood-corpuscles does not bear a direct ratio to the apparent health of the individual*. The hæmoglobin usually shows a *relative* increase. The number of white blood-corpuscles may be increased or diminished. The blood-plates are decreased in number. *Poikilocytosis* frequently occurs, but is not pathognomonic of pernicious anæmia. *Nummulation is imperfect or absent. The red blood-corpuscles are increased in size.* Shattuck and Cabot, found a red blood-corpuscle measuring 17 by 19.6 μ. The normal is about 7.5 μ. The nucleated red blood-corpuscles are characteristic

in the *predominance of megaloblasts*, and atypical corpuscles, and the diminution of normoblasts. The number of nucleated red blood-corpuscles may vary widely in the same case from time to time. *Polychromatophilic*, nucleated red blood-corpuscles, the protoplasm of which shows an affinity for Ehrlich's tri-color mixture, are found in pernicious anæmia more frequently than in other anæmias.

The *leukocytes* are diminished in number, but there is a *relative* increase in the number of the small mononuclear leukocytes. There is a small number of myelocytes, which occur in large numbers in splenic myelogenous leukæmia, and are occasionally found in chlorosis and the secondary anæmias.

The **diagnosis** is made by an examination of the blood. Secondary anæmias should be excluded. Every effort should be made to find the cause.

The **prognosis** is grave, but not necessarily fatal. The duration of life is usually about one or two years, rarely three or four years. Hope lies largely in the discovery of the cause, when the case becomes one of secondary anæmia rather than of pernicious anæmia.

Pernicious anæmia—treatment is largely symptomatic. Fowler's solution of arsenic, beginning with gtt. ij doses and increasing to tolerance, deserves a trial.

THE SECONDARY ANÆMIAS.

Secondary anæmia is found especially in cases of gastric ulcer, menorrhagia and metrorrhagia, hemorrhoids, hæmophilia, and traumatism; poisoning by lead and arsenic; malaria, syphilis, tuberculosis, leprosy, typhoid fever, septicæmia, Bright's disease, cirrhosis of the liver, and malignant disease.

Symptomatology: The individual is pale and suffers from muscular weakness, dyspnœa, vertigo, syncope, anorexia, vomiting, and emaciation.

The **diagnosis** has to do chiefly with the discovery of the cause, upon which the **prognosis** depends.

The **treatment**, aside from address to the cause, consists largely in the administration of iron and arsenic, and an abundance of nutritious food. Persistent cases may be benefited by a change of residence to a higher altitude.

LEUKOCYTOSIS.

In **leukocytosis** there is an increase in the number of leukocytes in the peripheral blood. The average number of white blood-corpuscles to the cubic millimetre of blood varies normally from 5000 to 10,000; in leukocytosis the number may reach 70,000 or more. The proportion of white to red blood-corpuscles, which normally varies from 1 : 400 to 1 : 1000, may reach in leukocytosis 1 : 50, or even 1 : 5 (Litten) in the death agony.

Leukocytosis **occurs normally**, or physiologically, in the new-born; after the digestion of proteids; during increased blood-pressure after exercise, massage, electricity and cold baths; especially during the later months of pregnancy; during the puerperium, gradually decreasing after the first day; and in the moribund state, and just before death.

Leukocytosis **occurs abnormally**, or pathologically, after hemorrhage, corresponding in degree with the anæmia produced rather than with the amount of blood lost; and in the infectious diseases. Perhaps the most marked leukocytosis has been found in the *bubonic plague*, in which 200,000 has been observed (Aoyoma).

Leukocytosis occurs in some cases of *diabetes;* in the so-called uric-acid diathesis; in cases of poisoning by illuminating-gas; after injections of ergotin and tuberculin or of the normal saline solution; during and after ether-narcosis; after the internal use of the salicylates; and in cases of malignant disease, especially when of rapid growth. Malignant disease may interfere with the ingestion of food, and thereby cause a decrease in the number of leukocytes.

The following diseases are marked by an **absence of leukocytosis**: Pure infections by the typhoid bacillus or tubercle bacillus, although typhoid fever and tuberculosis frequently show leukocytosis, due to the secondary septicæmia in these diseases; malaria, measles, leprosy, and intestinal obstruction, when not of a malignant character, and probably in influenza and cystitis.

Treatment should address the underlying disease.

LEUKÆMIA (**Leukocythemia**).

Leukæmia is distinguished by a *reduction of both red and white blood-corpuscles*, a relative increase of the white blood-corpuscles, and the general signs of anæmia.

The disease may be conveniently *divided into:* (1) *Splenic-myelogenous leukæmia*, in which there is an increased number of myelocytes, corpuscles which are believed to originate in the bone-marrow, with little alteration in the size of the spleen or lymphatic glands; and (2) *Lymphatic leukæmia*, in which the lymphatic glands show enlargement, with an increase in the number of leukocytes and a relative increase of the small mononuclear leukocytes as compared with the other white blood-corpuscles. Mixed forms are common.

The **etiology** is obscure. Most cases occur in men in middle life. *Leukæmia* is believed by many to be an infectious disease. Such an opinion is supported by the observation of Obrastow. An attendant who had charge of a case of lymphatic leukæmia and came into intimate contact with the case began to show the symptoms of the disease forty days later. The case in the attendant ran a course similar to that in the first patient. Both cases were fatal.

Transitions from leukæmia to pernicious anæmia, and *vice versa*, and from Hodgkin's disease to leukæmia, have been reported.

Among the conditions to which leukæmia has been attributed are malaria, syphilis, pregnancy, parturition, the climacteric, mental strain, traumatism, and heredity. Leukæmia is probably not caused directly by any of these conditions.

Leukæmia—symptomatology: There are *the usual symptoms of anæmia*. The *skin and mucous membranes are pale*, the patient complains of *palpitation of the heart*, *early fatigue*, shortness of breath on slight effort, and *hemorrhage*, especially *epistaxis*. Hemorrhage in the brain may be fatal; bleeding may also occur in the subcutaneous tissue, in the stomach, or in the bladder. Hemorrhage is most common in acute lymphatic leukæmia, occurring most frequently from the gums. Frequently there is diarrhœa, which is obstinate to treatment. The disease affects especially the spleen, or the lymphatic

glands, or the bone-marrow. The spleen may push the heart upward and interfere with its action. The liver is often enlarged. Dropsy is not uncommon. There is often fever, apparently without cause.

The **blood** is *normal in color* or somewhat pale, but *is not so fluid as normal blood*. There is usually a *dimunition of the hæmoglobin*. *Splenic-myelogenous leukæmia* shows an *enormous increase in the number of leukocytes*, averaging about 350,000 ; and also a large number of *myelocytes*. Myelocytes may be present in pernicious anæmia, but are much more numerous in leukæmia, averaging 37 per cent., or 80,000 per cubic millimetre. The polymorphonuclear cells show a *relative* decrease, but absolute increase in number. Lymphocytes and large mononuclear cells are present in small numbers. There may be a slight increase in the eosinophile cells.

In *lymphatic leukæmia* about nine-tenths of the white blood-corpuscles are *small lymphocytes*. A few myelocytes are usually present.

Diagnosis: Affection of the lymphatics, spleen, or marrow of the bones, in a patient with the appearance and symptoms of anæmia points strongly to leukæmia. An absolute diagnosis can be made by an examination of the blood, whereby the different varieties of the disease may also be recognized.

The **prognosis** is bad. Most cases die within two years, and only very rarely does life extend beyond four years. As in pernicious anæmia, about the only hope is that the cause may be found.

Leukæmia—treatment is symptomatic. The use of large doses of quinine or of Fowler's solution sometimes is beneficial. A diet made up chiefly of carbohydrates, and containing little of the proteids, has been recommended, since it has been proven that the proteids are poorly assimilated.

PSEUDOLEUKÆMIA (Hodgkin's Disease).

Pseudoleukæmia bears a strong resemblance to leukæmia. Transition-cases have been reported. Pseudoleukæmia is marked by enlargement of the spleen or lymphatic glands or

both; sometimes with enlargement of the lymphatics in various parts of the body.

The **etiology** is obscure. Many observers believe the disease to be due to an infection. Pseudoleukæmia occurs most frequently in men under forty.

Symptomatology: Aside from the *symptoms of anæmia* pseudoleukæmia is distinguished by the presence of *pressure-symptoms*, due to enlargements of the lymphatics or lymphatic glands.

Examination of the **blood** reveals as a rule no increase of the white blood-corpuscles. Of course, an increase of the white blood-corpuscles may appear as a coincidence, and not infrequently is present in cases of suppuration of an enlarged gland; but the leukocytosis is never marked; nor is there much degradation of the red blood-corpuscles, except toward the end of the disease, when they may number only 2,000,000. The reduction of the amount of hæmoglobin goes along with the reduction in the number of the red blood-corpuscles. There may be an increase of the lymphocytes or of the large mononuclear leukocytes. Myelocytes are sometimes present in small numbers, and occasionally normoblasts may be found.

The **diagnosis** depends upon the symptoms of anæmia, the pressure-symptoms, examination of the blood, and the exclusion of tubercular adenitis and syphilis. When the spleen alone is affected, malaria, leukæmia, rickets, and amyloid disease must be excluded.

The **prognosis** is grave. Acute cases may run their course in a few weeks. As a rule the course is not so rapid as in leukæmia. Usually the disease lasts for a number of years. Hope lies largely in finding the cause. Occasionally cases recover.

Treatment: The best single remedy is arsenic, which should be given both internally and hypodermatically. Further treatment is symptomatic.

THE HEMORRHAGIC DIATHESIS.

Definition: A disposition to hemorrhage upon slight injury is sometimes inherited. More frequently the condition is ac-

quired as a sequel to some infection, especially typhoid fever or smallpox; less frequently yellow fever, septicæmia, or diphtheria. The hemorrhage may come from the capillaries, *per diapedesin*, or from the larger vessels, *per rhexin*. Bleeding occurs most frequently from the nose and intestine; less frequently from the mouth, lungs, bladder, uterus, etc. Trivial insult may cause hemorrhage from any surface or into serous membranes and internal organs (brain).

Prognosis: Grave.

Treatment: *Epistaxis* may be arrested by plugging the nares. In general, hemorrhage may be controlled by rest, ice, tampons, and the internal use of opium, atropine, acetate of lead, digitalis, and the subcutaneous use of ergotin and sclerotinic acid. Further treatment is the same as for anæmia.

Hæmatidrosis (sweating of blood): A rare condition in which blood-corpuscles escape through the ducts of the sweat-glands in the presence of an unbroken skin.

PURPURA.

Definition: A class of affections characterized by the extravasation of blood into the skin.

Symptomatic purpura may be caused by the infections, septicæmia, and especially malignant endocarditis. Typhus fever, measles, scarlet fever, and smallpox are characterized by a purpuric rash. Toxic causes of purpura are snake-bites and occasionally certain drugs: copaiba, quinine, belladonna, mercury, ergot, and the iodides. Sometimes purpura appears in cancer, tuberculosis, Hodgkin's disease, Bright's disease, scurvy, and in old age.

So-called **myelopathic purpura** may appear in locomotor ataxia. Purpura may also be found in acute myelitis, transverse myelitis, occasionally in neuralgia, and the stigmata of hysteria.

Sometimes **purpura** occurs in cases of **venous stasis**, such as may occur in the paroxysms of whooping-cough and in epilepsy.

Arthritic purpura is characterized by the affection of joints. In *purpura simplex* the process is limited to a portion of the

body, usually the lower extremities, either with or without involvement of the joints. There is often an associate diarrhœa.

Purpura rheumatica, Schönlein's disease, is characterized by the affection of a number of joints and an eruption. *Purpura urticans* is a combination of wheals and purpura. In *pemphigoid purpura* there is an associated vesication. Sometimes there is œdema, constituting the condition known as febrile purpuric œdema. Schönlein's disease is characterized by multiple arthritis, purpura, and urticaria.

Henoch's purpura usually occurs in children and is characterized by numerous relapses and recurrences, cutaneous lesions, affection of joints, hemorrhages into the mucous membranes, and gastro-intestinal crises: pain, vomiting, and diarrhœa. Not all these symptoms are necessarily present in every case.

Purpura hæmorrhagica includes the cases of very severe purpura with hemorrhages from the mucous membranes. Sometimes cases prove fatal within a day, *purpura fulminans.* Death may occur before there is hemorrhage. Favorable cases of purpura hæmorrhagica recover in ten days to two weeks. The diagnosis calls for the exclusion of scurvy, and the recognition of smallpox and measles.

Treatment: Any apparent cause, such as may usually be found in symptomatic purpura, should be properly treated. For further treatment see the Treatment of the Hemorrhagic Diathesis.

HÆMOPHILIA (An Hereditary Hemorrhagic Diathesis).

Etiology: The disease is usually transmitted through a mother who is not herself affected, but is the daughter of a bleeder. The great majority of cases occur in males, as a rule within the first two years of life. The skin is usually fine and soft, and the individuals appear perfectly healthy. The cause of the condition is unknown.

Symptomatology: Slight lesions are followed by excessive bleeding. Usually the condition is first recognized as an epistaxis; sometimes by hemorrhage from the mouth, stomach,

bowels, urethra, lungs; more rarely from the skin of the head, tongue, finger-tips, tear-papilla, eyelids, external ear, vulva, navel, or scrotum. Even so slight an operation as the extraction of a tooth may prove fatal. Traumatisms that do not cause lesion of the skin or mucous membrane may be followed by the formation of petechiæ or even large hæmatomata. There is often arthritis, involving especially the large joints.

Prognosis: The outlook is grave, although individuals usually do not die from the first hemorrhage. The disease may be persistent even for years, so that the individual exceptionally may reach an advanced age.

Treatment: As prophylactic measures, individuals known to be *bleeders* or from suspicious stock, should be guarded from injury and not subjected to operations. The daughters in bleeder families, even though apparently unaffected, may transmit the disease to their descendents, without themselves showing any evidence of hæmophilia.

When bleeding occurs the usual remedies for the control of hemorrhage should be tried. As styptics, a solution of fibrin-ferment and sodium chloride, the application of fresh blood, and the use of a 5 per cent. solution of gelatin, have been recommended.

Further treatment is addressed to the general condition of the patient, with exercise in the open air, nutritious food, and the use of tonics, especially iron and cod-liver oil.

SCURVY (Scorbutus).

Definition: A disease characterized by hemorrhage, spongy gums, cachexia, and marasmus.

Etiology: The disease seems to depend upon an imperfect food-supply, especially the absence of those elements of food supplied by fresh vegetables. Many observers believe the true cause to be some micro-organism. Others would attribute the disease to a decreased alkalinity of the blood. Probably both views are correct.

Symptomatology: The onset is insidious. There are *progressive loss of strength and weight*, and the anæmia and depression of spirits characteristic of *cachexia*. *The gums are*

swollen and bleed easily, become spongy, ulcerated, necrosed, and covered with fœtid débris. The affection of the gums is confined to the region of the teeth. The breath is offensive. Epistaxis is frequent. Soon there is the picture of a general *hemorrhagic diathesis.*

Diagnosis: The recognition of scurvy is easy in the presence of an epidemic. Isolated cases may be recognized by knowledge of the character of the food used by the individual, in the presence of characteristic symptoms, and the improvement of the symptoms following the use of proper food.

Prognosis: Much depends upon the strength of the patient, the stage of the disease, and the ability to secure proper food.

Prophylaxis: The disease may be prevented by the use of fresh food, especially vegetables.

Scurvy—treatment: At first the juice of lemons or oranges may be given; later, apples, lettuce, potatoes, cabbage, watercress, sauer-kraut, spinach, onions, dandelions, and other fruits, vegetables, and greens may be added. The mouth should be cleaned and kept clean. Various antiseptic and astringent washes are recommended: peroxide of hydrogen, solutions of creolin, permanganate of potassium, and dilute carbolic acid. The gums may be treated with a solution of nitrate of silver. Constipation is best relieved by enemata.

ADDISON'S DISEASE (**Morbus Addisonii**; **Bronze-skin Disease**).

Definition: An affection of the suprarenal capsules, characterized by pigmentation of the skin, progressive anæmia, cachexia, gastro-intestinal catarrh, depressed circulation, and marasmus.

Etiology: The disease is rare in this country. Males are most frequently attacked. The most common lesion is tuberculosis of the suprarenal capsules. The disease depends upon a loss of function of these bodies. Carcinoma of the suprarenal capsules is rare. Traumatism seems to play a *rôle* in some cases.

Addison's disease—symptomatology: Usually the onset is insidious, with *anæmia and general debility.* The action of the heart is feeble. There are *symptoms of gastro-intestinal*

catarrh, nausea, and vomiting, later pain and retraction of the abdomen, sometimes severe anorexia, and at times diarrhœa. The most characteristic lesion is the *bronze pigmentation of the skin*, which may vary in color from light yellow to brown or even black. The pigmentation occurs first where there is normally a deposit of pigment, around the nipples and genitals; or where there is some irritation of the skin, as about the waist-band. The mucous membranes of the mouth, conjunctivæ, and vagina show pigmentation. There may be patches of pigment in the serous membranes. Usually the bronze color is first observed on the face and hands. The pigmentation may be diffuse. There is degeneration of the suprarenal bodies.

Diagnosis: The recognition of the disease depends upon the presence of the characteristic pigmentation, progressive cachexia, and marasmus.

Differential diagnosis has to do chiefly with the separation from abdominal tumors, pregnancy, disease of the liver, pediculosis, argyria; more rarely exophthalmic goitre, melanotic cancer, scleroderma, ulcer or dilatation of the stomach, and a free eruption of small black comedones.

Prognosis: Unfavorable. Rapid cases may reach a fatal termination in a few weeks. Protracted cases may last a number of years, sometimes with periods of improvement lasting for months, rarely with complete recovery. The outlook is usually best in the cases with most marked pigmentation.

Addison's disease—treatment: The suprarenal capsule may be administered raw, partially cooked, or in the form of the glycerin extract or the dried extract. In a collection of 48 cases treated in this manner, 22 were improved and 6 were reported cured (Kinnicutt). Further treatment is symptomatic (see Anæmia).

LITHÆMIA (Uric-acidemia; Uricæmia; Lithuria; Lithic-acid Diathesis; Uric-acid Diathesis; American Gout).

Definition: An excess of lithic (uric) acid in the blood with the production of symptoms, especially on the part of the nervous and digestive systems.

Etiology: Lithæmia occurs chiefly in individuals who are subjected to mental strain, worry, and anxiety, who eat too much, drink too little, and suffer from lack of exercise. Tobacco and alcohol often seem to play a *rôle* in causation. Other factors are heredity, cold climate, and neurotic temperament.

Lithæmia—symptomatology: The more common *nervous symptoms* are neuralgia, headache, vertigo, hebetude, insomnia, restlessness, and hypochondriasis. On the part of the *digestive system* there are coated tongue, lost or capricious appetite, pyrosis, weight and oppression in the epigastrium, sometimes nausea, vomiting, and gastralgia. There are flatulence, constipation, offensive stools, hemorrhoids, sometimes hepatic tenderness. The *skin* may show pruritus, eczema, urticaria, and lichen. *Genito-urinary* symptoms are urethritis, cystitis, orchitis, epididymitis, vaginitis, and endometritis (Anders): inflammations that are caused by slight insult in the presence of lithæmia.

The **diagnosis** is made by the presence of a number of the above symptoms. True gout gives a family history of the disease, and presents tophi and distorted joints.

Prognosis: Usually good under proper treatment.

Lithæmia—treatment: The individual must change his habits of life and subject himself to less worry and excitement. He must eat less, especially of meats and rich food, and drink an abundance of pure water. The cause must be removed. Outdoor life and bathing are beneficial. In the way of medicines, sodium phosphate and salicylic acid are of value for their action respectively upon the liver and excretion of urea. Further treatment is symptomatic.

GOUT (Podagra).

Definition: A disease characterized by an excessive formation of uric acid; and the deposition of urate of sodium in the joints; marked by paroxysmal pain and deformity of the joints, with affection of the heart and kidneys, and marasmus.

Etiology: There is a disturbance in metabolism, with ex-

cessive formation of uric acid. Cases have been observed in infancy; but, as a rule, the disease occurs late in life. Heredity seems to play a rôle in more than half the cases. Gout shows a preference for males. Rich food, the ingestion of alcohol, especially beer and ale, are the most prominent factors in etiology. The disease often occurs in cases of chronic plumbism.

Gout—symptomatology: The onset of the disease is usually sudden. The attack comes on often late in the night, with *pain* of greater or less severity, amounting at first sometimes only to a feeling of uneasiness, *localized in a joint, usually the joint of the big toe.* The pain becomes excruciating. The attack ceases in the course of an hour or two. The individual resumes his sleep; and in the morning the *joint is found to be red, swollen, tender,* and the movements limited. Such attacks come on in the midst of apparent health, more often following dyspepsia. The attack may be repeated the following night, or after a much longer interval, a month or year, depending upon the habits of the individual, especially with regard to the diet.

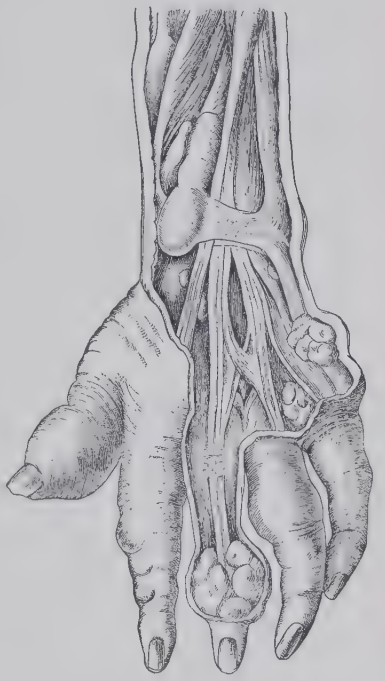
Chronic cases show *characteristic deformities* of the joints, due to the deposit of the urate of sodium. There are *chronic gastric catarrh, arterio-sclerosis, and affection of the heart and kidneys.* The enlarged joints may show ulceration, with the discharge of a substance composed largely of urate of sodium. The enlargements at first show fluctuation, later present a doughy sensation, and still later become hard tophi.

Gout—diagnosis: Usually there are dyspepsia and other evidence of over-indulgence in rich food and alcohol, especially the malt preparations, beer and ale. The polyarthritis usually, but not invariably, begins in the joint of the big toe. The inflammation remains fixed in the joints affected, which, together with the overlying tendons, become characteristically deformed through the deposit of urate of sodium in tophi (Fig. 37). Gout prefers the small joints. Males are most frequently affected. Examination of the urine shows the elimination of little or no uric acid during the paroxysm; during the interval the excretion of uric acid is greatly increased as a rule. Sometimes gout finds expression in con-

junctivitis, iritis, corneal ulcer; or the deposit of tophi in the ear, nose, eyelids, and larynx.

Gout should be *differentiated* from rheumatism, especially from arthritis deformans, in which the disease usually begins in the hands and more frequently involves the larger joints. There is usually little or no fever in gout.

Fig. 37.



Tophi in joints and tendons.

Prognosis: Good in acute gout. Chronic gout may take life through disease of the kidneys, heart, or brain.

Gout — treatment: Most important is the regulation of the life of the individual, especially as to diet and exercise. The use of artificial alkaline mineral water, or better a sojourn at the springs, may sometimes suffice even without the use of drugs. The waters usually recommended are Vichy, Carlsbad, and the lithiated waters. The most popular springs are those of Carlsbad, Homberg, Wildbad, in Germany; Contrexeville and Aix-les-Bains, France; Bath and Buxton, in England; and Saratoga, Bedford, and the White Sulphur Springs in this country.

In the way of *drugs*, most may be accomplished with the wine of colchicum, piperazin, and the salicylates. The limb should be wrapped in cotton-wool. The local application of hot air may be tried. The bowels should be kept open. Calomel is often of value early in the disease. Occasionally cases may be benefited by the iodides. Further treatment is symptomatic.

ARTHRITIS DEFORMANS (Nodular Rheumatism).

Definition: A chronic disease of obscure etiology, characterized by progressive, symmetrical deformity of the joints.

Etiology: Many observers believe the disease to be a chronic infection; others that it is of nervous origin. Most cases are found between thirty and fifty years. The great majority of cases occur in women. Heredity sometimes seems to play a *rôle*. There is a history of gout oftener than of true rheumatism. Exposure to cold, wet, damp, errors in diet, depressing mental emotions, prolonged sorrow, grief or dejection, are prominent factors in causation.

Arthritis deformans—symptomatology: Usually the onset is insidious. *Affected joints first become stiff, especially in the morning*, and tender, and later show *characteristic deformity*. The joints of the hands and fingers are usually first attacked. The fingers are flexed upon the hand and point toward the ulna; *the thumb is not affected*. When the disease attacks the foot, the big toe is first involved. The joints become locked, so that in cases of extensive involvement of the joints the patient may become immovably fixed in the position usually occupied. The disease is marked by great deformity. With exacerbations and abatements *the disease is progressive*. There are *impairment of the appetite and digestion* and *constipation*, largely due to lack of exercise. The patients become *irritable and hypochondriacal*. *The muscles undergo atrophy.*

Diagnosis: The only difficulty is offered early in the course of the disease. The disease prefers the female sex, and is comparatively rare under twenty. Permanent deformities are produced in the joints affected. Fever is usually absent. The disease is polyarticular and shows preference for the small joints. The hands and fingers, but not the thumbs, are usually first involved.

Prognosis: The disease does not seem to shorten life. It is chronic, and may be relieved, but not cured.

Arthritis deformans—treatment: The pain may be relieved by hydrotherapy and massage, which also assist the nutrition of the muscles. Electricity is sometimes of value. Arsenic probably does good as a tonic. Iron may be indicated by

anæmia. Iodine, best in the tincture, gtt. x, or the iodide of potassium or sodium, given in milk, is recommended. Salipyrin, salol, and the salicylates, and phenacetin may be advantageously used during the acute exacerbations. Massage and the local application of hot air sometimes produce good results. Blisters are of value, especially in chronic cases. Often a change of climate is advisable.

RICKETS (Rachitis).

Definition: A disease of infancy and childhood, characterized by gastro-intestinal disturbances, bronchial catarrh, impaired nutrition, and changes in the bones.

Etiology: Some observers attribute the disease to a chronic infection. An essential factor seems to be a faulty diet, especially one deficient in animal fat and proteid (Cheadle). The disease is most frequent from seven months to seven years, rare under six months, and in exceptional cases appears as late as the ninth to the twelfth year.

Rickets—symptomatology: There is early *gastro-intestinal disturbance*, marked by *anorexia, and diarrhœa or constipation.* There are *bronchial catarrh and cough.* The child becomes restless at night. *Sweating* occurs, often without apparent cause. There is *general soreness*, especially sensitiveness of the body. Sometimes cases are announced suddenly by a *spasm*, especially by laryngo-spasm. There is slight fever.

The bones show characteristic deformities. Nodules may be observed at the junction of the ribs and costal cartilages, forming the *rickety rosary.* The sternum projects, to form the *pigeon-* or *chicken-breast. The upper part of the head is large*, compared with the face and body of the child. Often *softened spots* may be made out over the bones of the skull, especially the occipital bone. *The fontanelles do not close early. The top of the head is more flat than normally.* The frontal eminences are prominent. The *teeth appear late* and often are ill formed. *The child grows slowly;* usually the stature is below the normal. *The bones are soft;* hence, the frequency of *bow-legs. The abdomen is usually large and distended.* The bones easily suffer fracture, especially the green-stick fracture.

Most dwarfs are rickety. Deformity of the pelvis is often caused by rickets.

Diagnosis: The disease is recognized by the characteristic changes in the bones.

Prognosis: Good. Fatalities depend upon complications.

Rickets—treatment: Malnutrition may be avoided by attention to the diet of the child. In cases of rickets the diet should include some fruit, such as lemon- or orange-juice. The child should be placed under good hygienic surroundings, receive plenty of pure, fresh air and sunshine, and a daily warm bath. When the bones are soft the child should not be permitted to remain in one position too long.

Of drugs, phosphorus has the best reputation. The remedy is given in cod-liver oil, 1 : 16, a teaspoonful after meals, three times a day. The syrup of the iodide of iron may be added. Further treatment is symptomatic.

OSTEOMALACIA.

The disease is **characterized** by softening and consequent deformity of the bones.

Etiology: Numerous micro-organisms have been found in osteomalacia. The bones contain an excess of lactic acid; but this is sometimes found in the absence of osteomalacia. Some observers believe the condition due to a disturbance of the centres that preside over nutrition.

Symptomatology: The bones become tender, and pain is often first noticed in the pelvis, spine, and thighs, especially during the latter part of gestation. The muscles of the thighs and pelvis may suffer weakness, pain, and spasm. The affected bones become soft and show deformity. The stature of the individual is diminished (Strümpell). Increased knee-jerk and ankle-clonus are prominent symptoms. The disease is confined almost exclusively to the female sex. The affected women are usually above the average in fertility (Eisenhart). Abortion is frequent. The blood shows diminished alkalescence (von Jaksch), and contains myelocytes and an increased number of eosinophilic cells (Musser).

Diagnosis: The disease may be suspected in the presence of

pain in the pelvis, spine, and thighs, in women, especially when the pain recurs in succeeding pregnancies. Usually the height is diminished. The softening and deformity of the affected bones are characteristic.

Differential diagnosis has to do chiefly with rheumatism, spinal disease, peripheral neuritis, and diffuse infiltration of bones with malignant growths.

Prognosis: The disease is usually progressive; but may frequently be brought under control by proper treatment.

Osteomalacia—treatment: The use of phosphorus (Sternberg) and cod-liver oil (Trousseau) has given good results. In the way of surgery, ovariotomy and Porro's operation have both been followed by recovery in a large number of instances.

OBESITY (Polysarcia; Corpulence).

Definition: An excessive general deposit of fat.

Etiology: Obesity occurs especially after forty. Sometimes the condition appears in young persons. The chief causes are excessive eating and sleeping and lack of exercise. Women are most frequently affected.

Symptomatology: The development of fat may be termed obesity only when it accumulates to such a degree as to interfere with the comfort and health of the individual.

Treatment: The individual should eat less, take more exercise, best in the open air, and less sleep. The ingestion of fluids should be limited. Probably the best dietaries are those given by Banting, Ebstein, and Oertel. Banting reduces the amount of food and drink, and excludes the fats and carbohydrates. Ebstein permits the use of fats and excludes the carbohydrates. Oertel limits the fat, and permits the use of albumin and starch, and advises systematic exercise for the purpose of increasing the strength of the heart. Skimmed milk and massage are recommended by Weir Mitchell. Warm baths are of value. Sometimes good results may be secured by the use of the thyroid extract.

DIABETES MELLITUS.

Definition: A disease characterized by polyuria, glycosuria, and progressive impairment of health and strength.

History: Celsus recognized the polyuria and emaciation. Glycosuria was suspected, from the sweet taste of the urine, by the Arabian physician Susruta, in the seventh century, and became generally known when re-discovered by Thomas Willis (1674). The sweet taste was proven to depend upon sugar by Matthew Dobson (1775). Cowley (1778) separated the sugar by evaporation. Rollo (1798) introduced the meat-diet and the use of opium in the treatment of diabetes. Chevreuil (1815) observed that the sugar of diabetic urine is the same as grape-sugar. Tiedemann and Gmelin discovered the formation of sugar from starch during digestion. Ambrosiani (1835) found sugar in the blood of diabetic patients. This Mialhe believed to be due to diminished alkalescence, caused by suppression of the secretion of the skin. Stosh (1828) observed diabetic coma. Bernard (1856) produced glycosuria by puncture of the floor of the fourth ventricle. Marchel (1852) recognized diabetic gangrene. Frerichs and Von Recklinghausen (1866) observed disease of the pancreas frequently in diabetes; and later Mering and Minkowsky found diabetes to follow extirpation of the pancreas.

Etiology: The disease is found more frequently in some places than others. Certain races, for instance, the Israelites, are frequently affected; while others, especially the negro, seldom show the disease. Most cases occur in the male sex, and at from twenty to fifty years of age, especially between thirty and forty. Only about one-fourth of the cases are found in the female sex. Age and infancy are not exempt. Sometimes heredity seems to play a *rôle*. Diabetics often show disease of the kidneys, blood, nervous system, and pancreas; but diabetes may occur in the absence of disease of these organs.

Anxiety, luxury, alcohol, and obesity are prominent etiological factors. Most cases show arterio-sclerosis. Syphilis probably plays a minor *rôle*. Diabetes sometimes appears after trauma, in the course of or after nervous diseases, emo-

tional disturbances, and the infectious diseases. The belief has been advanced that diabetes is contagious.

Diabetes *follows* total extirpation of the pancreas; but if more than one-tenth of the organ be left, diabetes does not result. It has been demonstrated experimentally that removal of the pancreas is not followed by diabetes if at the same time the medulla or spinal cord be divided in the region of the upper cervical vertebra. Also, experimental removal of the pancreas is not followed by diabetes if the liver be removed at the same time.

Diabetes mellitus—symptomatology: The *onset* is insidious, with anorexia, nausea, headache, and insomnia, symptoms usually ascribed to dyspepsia or neurasthenia. There are *thirst and polyuria*, the urine discharged in the twenty-four hours amounting to more than three pints, usually four to eight pints, sometimes more.

The *urine* is light colored and foams readily. The reaction is acid; the *specific gravity high*, 1030–1040, rarely lower than 1020, nor higher than 1050. Trousseau reported as high as 1074. There is *glycosuria*, the amount of sugar varying from 2 per cent. or less in mild cases, to as high as 10 per cent. Rarely there is pneumaturia, gas discharged with the urine. Other abnormal ingredients found in diabetic urine are acetone, acetic acid, ammonia, and oxybutyric acid. The proportion of cases showing albuminuria has been variously given by different observers. Frerichs found albuminuria in 5 per cent. of cases; Rokitansky, in 65 per cent. There is not often œdema. Sometimes there is cystitis. The urine may contain short hyaline casts.

The *appetite* is sometimes voracious and insatiable. Nevertheless there is *progressive impairment of health and strength*. As a rule there is constipation. Often there is impotence. The knee-jerk may be diminished or lost. The increased secretion of urine is in marked contrast with the lessened secretion of the skin. Often there is *pruritus*, especially pruritus vulvæ and *furunculosis*, sometimes phlegmonous inflammations and *gangrene*. Cataract is not infrequent.

Among the nervous symptoms are *headache*, neuralgia, paræsthesia, and *coma*. *Tuberculosis* is a frequent complication.

Diabetes mellitus—diagnosis: 1, impairment of health; 2, increased quantity of urine; and 3, the presence of sugar in the urine. Glycosuria may be detected by the following

Tests for Sugar:

Bremer's test: The specific gravity of the urine must not be less than 1015. To 10 c.c. of urine, at a temperature of 14° or 15° C., in a test-tube, add gr. $\frac{1}{30}-\frac{1}{40}$ of methyl-violet or ethylene-blue. Normal urine does not dissolve methyl-violet; diabetic urine dissolves the dye and assumes a deep violet or bluish-violet tint. When ethylene-blue is used, normal urine gives a green color, and diabetic urine a blue. The test gives a positive reaction when normal urine is diluted with water. Thus the Bremer test might be of value in life-insurance examinations, in the detection of fraud, when individuals are suspected of diluting the urine in order to give a lower specific gravity.

Moore's test: To a test-tube one-third full of urine an equal quantity of concentrated KHO is added and heat applied. Sugar is indicated by a brownish color.

Trommer's test: To equal quantities of urine and concentrated KHO add 1 per cent. solution of copper sulphate, drop by drop, under gentle heat, as long as the copper sulphate will dissolve. Sugar gives a yellowish or reddish precipitate before the boiling-point is reached.

Hain's test: Hain's fluid: copper sulphate, gr. xxx; glycerin, ℥ss; aquæ, ℥ss; liquor potassæ (U. S. P.), ℥v. Heat the test-fluid and add four or five drops of urine. Sugar gives a yellow or red, salmon-colored precipitate.

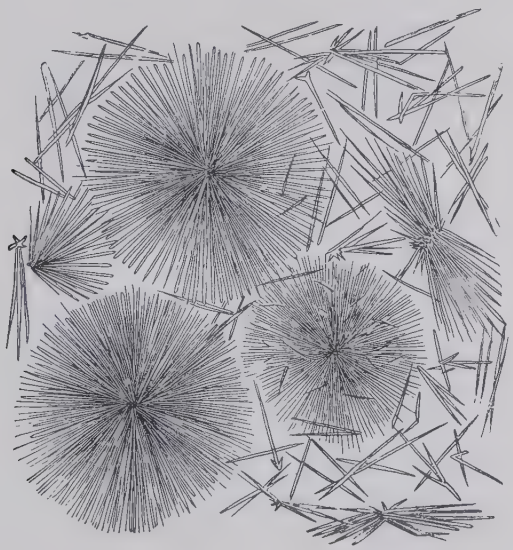
Bottger's test: To equal quantities of urine and KHO add bismuth subnitrate and apply heat. Sugar gives a black color. The test is useless in the presence of albumin. A better bismuth test is:

Nylander's test: Nylander's reagent: Rochelle salt, 4.0; 8 per cent. solution of NaHO, 106.0; add bismuth subnitrate to saturation.

Method: To ten parts of urine add one part of the reagent and boil two minutes. A black color indicates sugar.

Phenyl-hydrazin test: To 6–8 c.c. of urine add phenyl-hydrazin hydrochlorate (twice as much as will go on the point of a penknife) and pulverized acetate of sodium (three point-of-a-penknifefuls). Heat. If the reagent does not dissolve, add hot water. Cool. If sugar is present, there will be formed a yellow precipitate, which may appear amorphous to the naked eye, but under the microscope will be seen to consist of fine needles of phenyl-glucosazon arranged in stars (Fig. 38).

Fig. 38.



Crystals of phenyl-glucosazon (von Jaksch).

Fermentation-test: To 10 c.c. of urine add 1 gramme of commercial compressed yeast ($\frac{1}{16}$ Fleischmann cake). Shake until the yeast is dissolved. Place in a saccharometer and leave at the room-temperature for twenty-four hours. In the presence of sugar alcoholic fermentation causes the formation of carbonic acid gas, which gathers at the top of the saccharometer and causes the fluid to change its level. The percent-

age of sugar may be read off the scale. When the specific gravity is above 1022 the urine should be diluted from two to ten times, depending upon the height of the specific gravity, and the reading of the saccharometer should then be multiplied accordingly.

Roberts has observed that urine after fermentation is of lighter specific gravity than before, and that the difference in specific gravity is such that every degree lost is approximately equivalent to one grain of sugar (glucose). The test may conveniently be made by dissolving a cake of Fleischmann yeast in four ounces of urine, taken from the total quantity passed in the twenty-four hours, and placing in a pint bottle in a warm place for twenty-four hours. The bottle should be loosely corked, to permit the escape of the carbonic acid gas. Four ounces of the urine, tightly corked, are used as a control-specimen. At the end of twenty-four hours the specific gravity of the two specimens is taken; the difference represents the amount of sugar in grains, and this multiplied by 0.23 will give the approximate percentage of sugar.

Prognosis: As a rule, which is not invariable, the outlook depends upon the amount of sugar in the urine. Most cases are not curable, but usually the disease may be brought under such control that the patient may live to an advanced age. In general, the patients hold their fate in their own hands, for the outlook depends largely on the diet. Severe nervous symptoms, especially coma, are ominous.

Cases of acute diabetes, *diabetes acuta* and *acutissima*, may terminate in a few weeks or months. As a rule diabetes is chronic, lasting for a number of years or for life.

Diabetes mellitus—treatment: Most important is the *diet*. The following diet-list is given by Van Noorden:

8 o'clock, first breakfast.	3 ounces of ham. 1 cup of tea. 1 glass of cognac.
10.30 o'clock, second breakfast.	2 eggs, fried in $\frac{1}{3}$ ounce of butter.

12.30 o'clock, luncheon.	5 ounces of cold roast meat. Mayonnaise, made with the yolk of 1 egg and 1 spoonful of oil. Raw cucumber, with $\frac{1}{6}$ ounce of vinegar, 1 spoonful of oil, salt and pepper. $\frac{1}{2}$ ounce of Gorgonzola cheese. $\frac{1}{2}$ bottle of Moselle. 1 cup of coffee with tablespoonful of cream.
5 o'clock, tea.	1 cup of tea. 1 boiled egg. 1 glass of cognac.
7.30 o'clock, dinner.	1 cup of bouillon, with $\frac{1}{2}$ ounce of marrow. $2\frac{1}{2}$ ounces of boiled salmon. $\frac{1}{3}$ to $\frac{1}{2}$ pound of asparagus, with $\frac{2}{3}$ ounce of butter. 1 ounce of smoked ox tongue. 3 ounces of capon. Salad, with $\frac{1}{6}$ ounce of vinegar and 1 spoonful of oil. $\frac{1}{2}$ bottle of Burgundy.
10.30 o'clock, nightcap.	1 glass of cognac, with Seltzer water.

Of *drugs*, opium is one of the most valuable remedies, but may be used only a short time. Codein has much of the virtue of opium with fewer evils. Some cases respond well to benzosol, the benzoate of guaiacol, in five-grain capsules, one every four hours. Often good results may be obtained with Jambul, the Java plum, or in the form of the fluid extract, ♏ x, in powders of five to ten grains, gradually increased to gr. 75–150. Relief is sometimes afforded by salicylate of sodium, 5–10 grammes, or benzoic acid, 3–5 grammes. Coma may demand infusion of the normal salt solution (0.6 per cent. of chloride of sodium) into the rectum, under the skin, or in bad cases into the veins. Often good results follow lavage.

Gangrene calls for the intervention of surgery.

DIABETES INSIPIDUS.

Definition: A disease characterized by increased secretion of urine, polyuria, without glycosuria. The condition differs from a simple polyuria, such as may follow the ingestion of large quantities of fluid, chiefly in that there is impairment of the general health.

Etiology: The disease shows a preference for youth and for the male sex. Cases have been observed after traumatism, sunstroke, violent emotion, sometimes excessive drinking of cold water, or after a protracted spree, or during convalescence from the acute infections. Many believe the condition to be of nervous origin. Sometimes lesions of the medulla, tumors of the brain, meningitis, have been found in cases of diabetes insipidus. The disease is sometimes congenital. Weil reports twenty-three cases in four generations in a family of ninety-one members, which would seem to indicate a *rôle* played by heredity.

Symptomatology: *The urine is increased in quantity* to twenty, forty, or more pints in the twenty-four hours, and is of low specific gravity, 1001–1005, light in color, and contains little sediment. With the discharge of so much fluid there is thirst. There may be no impairment of the general health.

Diagnosis depends upon the discharge of a large quantity of urine of low specific gravity, without the presence of sugar. The polyuria of hysteria may be eliminated by the absence of other evidence of hysteria. Furthermore, hysterical polyuria is more transitory. Bright's disease may sometimes give a large quantity of light urine, but there is always some albuminuria, which is rare in diabetes insipidus.

Prognosis: The disease usually runs a chronic course. Medication seems to have little effect as a rule. Sometimes spontaneous recovery takes place. Cases have been known to persist as long as fifty years. Usually death results from some intercurrent malady.

Diabetes insipidus—treatment: Opium will diminish the amount of urine, but is of doubtful value because of the re-

mote evils attending its use. Thirst should be relieved by frequent rather than copious libations. Among the remedies recommended are valerian, the valerianate and lactate of zinc, ergot, ergotin, antipyrin, antifebrin, the salicylates, arsenic, strychnine, turpentine, and the bromides. Open-air exercise and the use of electricity are often of value.

CHAPTER VI.

DISEASES OF THE GENITO-URINARY ORGANS.

ALBUMINURIA.

The **presence of albumin in the urine**, abuminuria, is caused almost entirely by the transudation of blood, especially serum-albumin, from the bloodvessels into the tubules of the kidney. The exfoliation of a large number of epithelial cells may give rise to a trace of albumin in the urine.

Etiology: Albuminuria does not always depend upon disease of the kidneys. Small quantities of albumin may sometimes be found in the urine in pregnancy, after severe and prolonged exertion, and after the ingestion of large quantities of food, especially albuminous food, eggs, cheese, pastry, particularly when not properly digested, and when the individual indulges in exercise immediately after eating.

Albuminuria may result from some change in the composition of the blood, as in anæmia and some cases of puerperal eclampsia, without inflammation of the kidney. In some puerperal cases, and in chronic congestion of the kidney, albumin appears in the urine as the result of changes in the blood-pressure. Some cases of albuminuria are due to changes in the walls of the capillaries not of an inflammatory character.

More important and numerous are the cases of albuminuria due to disease of the kidney, inflammation of the walls of the capillaries, which thus more readily permit transudation. To this category belong the cases of *acute nephritis*, the severe forms of acute degeneration, acute congestion, and some cases of chronic nephritis with exudation.

Accidental albuminuria may be caused, outside of the kidney, by pyuria, hæmaturia, the escape into the urine of seminal or prostatic fluid, more rarely chyluria, and not infre-

quently by hemorrhage or transudation of serum from some part of the urinary tract below the kidneys. Albuminuria from the exfoliation of epithelial cells has been mentioned.

Tests for Albumin.

Heller's test: Cloudy urine should be filtered. Boil the urine and add concentrated nitric acid. Albumin gives a white precipitate. A similar precipitate may be obtained when patients are taking balsam, which, however, may be dissolved by alcohol. Phosphates are precipitated by heat and redissolved by the acid. Urates are dissolved by the heat.

Potassium-ferrocyanide test: Acidify the urine with acetic acid, and add 10 per cent. solution of potassium ferrocyanide. Albumin is precipitated.

Spiegler's test: Spiegler's test-fluid: corrosive sublimate, 40; tartaric acid, 20; white sugar, 100; and distilled water, 1000.

Method: A layer of urine is allowed to flow gently upon some of the test-fluid in a test-tube. Albumin is indicated by a white precipitate formed at the junction of the urine and the test-fluid.

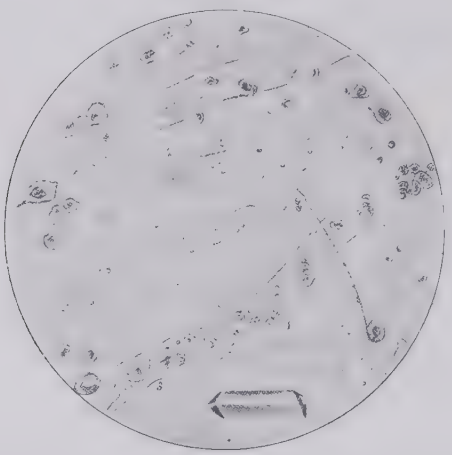
Heller's test precipitates *serum-albumin* (and albumose when cold). The *potassium-ferrocyanide* test precipitates *serum-albumin* and *albumose*. *Spiegler's test* precipitates *serum-albumin, albumose,* and *peptones.*

Peptonuria indicates suppuration somewhere in the body. Its determination may be of value sometimes when we can exclude scurvy, intestinal ulceration, and the puerperium. In obscure cases its absence indicates the absence of suppurative processes in the body. Thus it is a means of differentiation between suppurative and tubercular meningitis.

Casts.

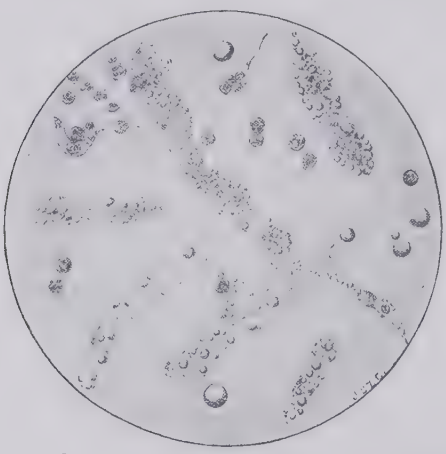
(1) **Epithelial casts** are composed partly or wholly of epithelial cells from the tubules of the kidney, and are indicative of a parenchymatous nephritis.

FIG. 39.



Hyaline casts from a case of acute nephritis. 1, plain hyaline cast; 2, granular deposit of hyaline casts; 3, cellular deposit (blood and epithelium).

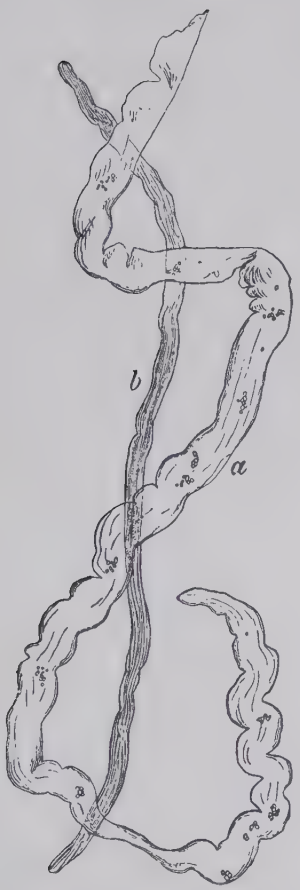
FIG. 40.



Fatty casts from a case of chronic parenchymatous nephritis.

(2) **Blood-casts**, composed of more or less perfect blood-

Fig. 42.



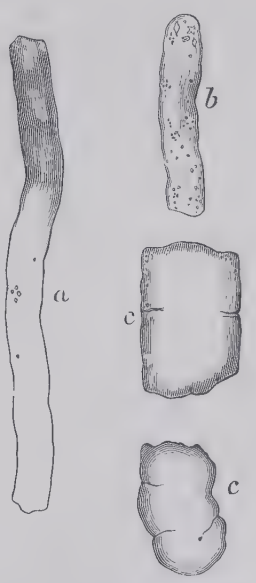
Cylindroids from the urine in congested kidneys (von Jaksch).

corpuscles, indicate hemorrhage from the kidney: (*a*) acute congestion of the kidney, (*b*) acute inflammation of the kidney, (*c*) infarction of the kidney.

(3) The constant presence of **pus-casts**, which are rare, may be due to multiple abscess of the kidney.

(4) **Casts composed of micro-**

Fig. 41.



Different forms of waxy casts (von Jaksch).

cocci may be discharged in cases of renal sepsis (embolism), suppurative nephritis, and pyelonephritis.

(5) **Granular casts** are found in the presence of degeneration of the renal epithelium.

(6) **Fatty casts** indicate fatty degeneration, such as may be present in the large white kidney, or in cases of poisoning by phosphorus, antimony, or iodoform.

(7) **Hyaline casts** point strongly to chronic interstitial nephritis.

(8) "**Waxy**" **casts** (broad hyaline casts) are found in amyloid degeneration of the kidney.

(9) **Cylindroids,** or streamers, which are not true casts, indicate irritation of the kidney. They are often present in lithæmia and oxaluria.

DROPSY.

Dropsy depends upon an increased transudation of blood-serum from the capillaries and diminished absorption by the lymphatics. Inflammatory exudation and passive dropsy may be caused by increased blood-pressure or some change in the blood, especially the injury caused by poisons (toxins) circulating in the blood.

Dropsy due to *disease of the kidneys* is expressed first as œdema of the lower eyelids and ankles; later, of the legs and serous cavities; and finally of the entire body—anasarca. A fatal termination may be caused by œdema of the glottis, lungs, or bronchi.

URÆMIA.

Uræmia is probably a misnomer, since the so-called uræmic symptoms do not always follow the injection of urea into the blood, nor is there always an increased amount of urea in uræmia.

The condition is *caused* by the circulation in the blood of some poison, possibly a toxin, that normally is excreted through the kidneys.

Uræmia—symptoms: 1, *headache*, hebetude, somnolence or insomnia, and anxiety, which may occur in cases of nephritis, either acute or chronic, and in cases of puerperal eclampsia, with or without nephritis; 2, *hemiplegia and aphasia*, separately or together in chronic nephritis or eclampsia, ascribed by some to an endarteritis; 3, *blindness*, amaurosis, which

comes on suddenly in puerperal eclampsia and sometimes in chronic nephritis; 4, *general epileptiform convulsions*, in puerperal eclampsia and in acute or chronic nephritis. Other symptoms are: muscular contractions, delirium, coma, vomiting, diarrhœa, fever, dyspnœa, and increased arterial tension, due to hypertrophy of the left ventricle.

PYURIA.

Pus in the urine, pyuria, may be recognized readily by microscopic examination of the urine. Sometimes pus is present in such quantities as to be obtrusive.

Etiology: A heavy deposit of pus in acid urine usually comes from pyelitis or chronic pyelonephritis. The constant discharge of muco-purulent urine is usually found in conjunction with bladder-symptoms. Such urine, in the absence of vesical symptoms, especially when accompanied by the discharge of blood, may come from pyelitis due to the presence of a calculus. The intermittent discharge of purulent urine usually indicates pyelitis.

Sometimes pus comes from outside the urinary tract. Fever will then accompany the accumulation of pus, to disappear upon its discharge. The origin of the suppuration may be recognized in some cases by the presence of pain and other symptoms.

Flakes or threads of pus usually come from the urethra, but may come from other parts of the urinary tract, as in cases of pyelitis, nephrolithiasis, tuberculosis of the bladder and prostate, and perivesical abscess.

Often the presence of epithelial cells, recognized upon microscopic examination of the urine, in cases of pyuria, will aid materially in determining the origin of the pus.

A careful examination, especially by palpation, should be made of the entire urinary tract. The use of the cystoscope is often of value. As a rule the source of the pus will be found in the pelvis of the kidney, the bladder, or the urethra. As stated, pus may come from without the urinary system—*e. g.*, perivesical abscess and hip-joint disease.

CHYLURIA.

The **presence of chyle** gives to the urine a milky appearance. Sometimes the urine is more or less colored by the admixture of blood. The amount of fat varies from 0.2 to 2 per cent., and may be dissolved by the addition of ether, whereupon the urine loses its milky appearance.

Microscopic examination of the urine reveals the presence of fat. The filaria sanguinis hominis, which is a common cause of chyluria, is found in the urine secreted during the day, as a rule, and in blood withdrawn during the night (see Filaria Sanguinis Hominis). The chyle and filaria probably gain entrance to the bladder through some communication between the dilated lacteal channels and the urinary tract.

HÆMATURIA.

Etiology: Hemorrhage may take place from any part of the urinary tract. Aside from traumatism, nephritis, and gonorrhœal prostatitis, hæmaturia is caused most frequently by stone in the bladder, tumors of the bladder, stone in the kidney, tuberculosis of the bladder, tuberculosis of the prostate, carcinoma of the kidney, cystitis, and enlarged prostate. Some cases are due to the distoma hæmatobium.

An effort should be made to determine the cause of the hæmaturia. Some knowledge may be gained by observing the color of the urine, the presence and shape of clots of blood, the time at which the blood is discharged in the stream of urine voided, the intimacy with which the blood is mixed with the urine, and the microscopic examination of the sediment.

A careful physical examination should be made of all accessible parts of the urinary tract, the kidneys, ureters, bladder, prostate, and testicle by palpation, and if necessary by the use of the endoscope and cystoscope.

Hæmaturia may occur in some of the infectious diseases, especially in measles, smallpox, typhus fever, septicæmia; cholera, malaria, scurvy, and in the hemorrhagic diathesis. Certain drugs, especially cantharides and turpentine, may cause hæmaturia.

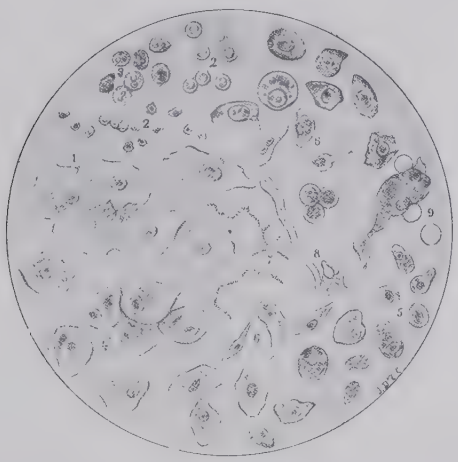
PYELITIS.

Pyelitis is an inflammation of the *pelvis* of the kidney.

Pyelonephritis is a conjoint inflammation of the substance and pelvis of the kidney. When suppuration extends to cause destruction of the kidney-substance and form a large abscess-cavity the condition is known as *pyonephrosis*. The involvement of surrounding tissues leads to *perinephritis*, *paranephritis*, and sometimes to the formation of abscess.

Inflammation of the pelvis of the kidney, *pyelitis*, may be *primary*, caused by the discharge of some irritating sub-

FIG. 43.



Cellular elements from the urine. 1, squamous epithelium; 2, red blood-corpuscles; 3, polynuclear leukocytes; 4, transitional cells; 5, epithelium from the kidneys; 6, epithelium from the pelvis of the kidney and the bladder; 7, micrococcus ureæ; 8, yeast-fungi.

stance through the kidney; or *secondary*, caused by infection travelling from below upward along the urinary passages. Thus, the cause may come *from above*, as in tuberculosis of the kidney; or the passage of *micro-organisms*, more especially the *toxins*, toxalbumins, of the infectious dis-

eases—typhus, typhoid fever, septicæmia, influenza, smallpox, scarlet fever, diphtheria, tuberculosis, or cholera; or the elimination of certain drugs—cantharides, turpentine, or copaiba.

The most frequent *local* cause of pyelitis is kidney-stone. Some cases depend upon traumatism.

The cause may come *from below*, from a cystitis, gonorrhœa, or the use of unclean instruments.

Pyelitis—symptomatology: There is dull *pain*, radiating from the kidney along the ureters to the bladder. More often there is a *feeling of tension and weight in the region of the kidney*. The *urine* contains *pus*, sometimes *blood* and *albumin*, and the tailed *epithelial cells* normally found in the pelvis of the kidney. The urine is acid in reaction, or becomes alkaline only when there is retention.

Diagnosis: There is *pyuria*, sometimes hæmaturia and albuminuria, and the discharge of characteristic epithelial cells. The presence of hebetude, the typhoid state, with chills and fever, would indicate pyelonephrosis.

The **prognosis** is serious, but depends largely upon the cause. Cases dependent upon cystitis or kidney-stone usually disappear upon the relief of these conditions. When due to the infections, much depends upon the nature of the infectious agent. The outlook is better after typhoid fever than after septicæmia.

Pyelitis—treatment: In the way of prevention, only clean instruments (catheters) should be used; kidney-stones should be removed; gonorrhœa properly treated. The kidney may be flushed by the free use of water, which is best administered in the form of alkaline mineral water. The patient should observe absolute rest in bed. The bladder may be washed with dilute solutions of creolin. The salicylates and methylene-blue may be given to limit or prevent bacterial activity. Other remedies highly recommended are quinine, dilute hydrochloric acid, creosote, turpentine, and the oils of copaiba and sandalwood. Sometimes resort must be made to nephrotomy or nephrectomy.

FLOATING KIDNEY (**Wandering Kidney**; **Movable Kidney**; **Ren Mobile**).

Undue mobility of the kidney is caused by the presence of a *mesonephron*, undue laxity of the abdominal walls, more often by compression by belts, corsets, and still more frequently, probably, by traumatism, violent concussions of the body.

Floating kidney—symptomatology: There may be no symptoms. The patient sometimes complains of the *symptoms of dyspepsia*, which are not relieved by the usual treatment of dyspepsia. There may be *pain*, *colic*, *abdominal dragging*, *sensations of displacement*, sometimes icterus, and symptoms of the most varied character. A *movable tumor* may be felt upon palpation; but the failure to find such a tumor does not necessarily exclude floating kidney. The kidney is usually sensitive.

Prognosis as to life is good. As a rule permanent relief may be secured only by appeal to surgery.

Treatment: Should satisfactory relief not be afforded by the use of an abdominal supporter, *nephrorrhaphy* (fixation of the kidney by suture) should be resorted to. *Nephrectomy* (removal of the kidney) may be necessary when the organ is diseased.

NEPHROLITHIASIS; **KIDNEY-STONE** (**Renal Calculus**; **Gravel**; **Sand**).

Over 99 per cent. of all urinary calculi originate in the kidneys. **Kidney-stones** may consist of uric acid or urates, oxalate of lime, less frequently of cystin, carbonate of lime, xanthin, or indigo; sometimes two or more of these substances in combination; and in the presence of suppuration, and decomposition of urine, there may be deposits of phosphate of lime and triple phosphate. Kidney-stones occur in all varieties of shape, and in size from so-called sand to over a thousand grammes in weight; and from one to over a thousand in number.

Etiology: The great majority of cases occur in males, most frequently from two to twelve years old. Association of

kidney-stone with gout has been frequently noted. Cases are often ascribed to an excess of uric acid in the blood, or a diminution of the biphosphate of sodium, a salt that holds the uric acid in solution. Phosphates are precipitated in an alkaline urine, the result of decomposition. *Nephrolithiasis* may occur at any age; calculi have been found in the kidneys of the new-born.

Some observers believe a prominent *rôle* in etiology is played by the mucus and possibly some colloid material secreted by the kidney.

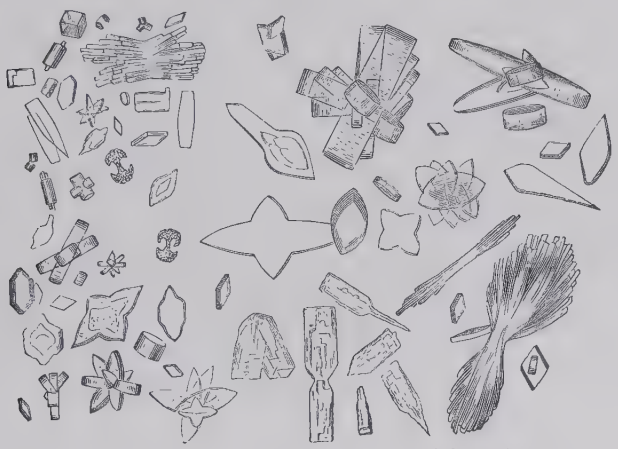
Symptomatology: There may be no symptoms. But usually the presence, especially the passage, of *kidney-stones* gives rise to distinct symptoms. The most common symptom is *pain, radiating from the kidney to the bladder*, aggravated by movement of the body, and usually increased by pressure over the kidney. The pain is irregular, occurs in *paroxysms*, and may be relieved only by large doses of opium or morphine or the use of anæsthesia. There is usually *hæmaturia*, often *pyuria*, and sometimes *albuminuria* even independently of the presence of blood. The *pain*, especially during the passage of a calculus through the ureter, is intense. With the paroxysm there are often rigor, vomiting, cramp, and profuse perspiration. Pain is often reflected to the groin, testicle, gluteal region, and inner side of the thigh and leg. Often there is retraction of the testicle. With the passage of the stone into the bladder the paroxysm suddenly ceases, possibly to leave the patient narcotized if much opium or morphine has been administered. The *urine* often contains *mucus* during the retention of a stone in the kidney.

Diagnosis: Sometimes the patient comes with the diagnosis already made by the passage of a stone, more often of the small particles known as sand, or with the history of having passed a calculus. Such specimens should be examined to determine the character of the stone.

The *most common* urinary calculi are the uric acid and phosphatic stones. When there is no history of the passage of a calculus the characteristic pain, the presence of blood and mucus, sometimes of pus in the urine, should lead to a microscopic examination of the urine, whereby crystals may be

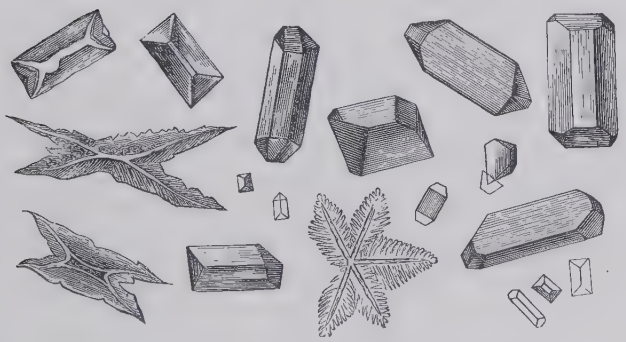
found, to reveal the presence and character of the stone. The condition should not be mistaken for the passage of uric acid

Fig. 44.



Various forms of uric-acid crystals (Finlayson).

Fig. 45.



Various forms of triple phosphate (Finlayson).

in gout, or of the débris from tubercular or cystic kidneys or hydatids of the kidney.

Prognosis should be guarded. Recovery is the rule. The

chief danger is rupture of the ureter and consequent peritonitis; or the formation of abscesses, with sinuses and fistulæ, and consequent marasmus; and amyloid degeneration in the various organs.

Kidney-stone—treatment: During the attack the pain should be relieved by moderate doses of morphine combined with atropine, round doses of chloral, the application of moist heat, and if necessary the use of an anæsthetic.

Curative treatment consists in thorough flushing of the kidneys by free libations of fluids, the mineral waters—Saratoga, Bethesda, Carlsbad, Contrexeville, Ems—and the lithiated waters, barley-water, or pure water. Piperazin, gr. v–xv, may be given in soda or Seltzer water three to five times a day. Gentle massage is sometimes of value. Upon failure of these means an appeal must be made to surgery. *Nephrolithotomy* or, in the presence of extensive disease of the kidney, *nephrectomy* may be indicated.

HYDRONEPHROSIS.

Etiology: When the escape of urine is prevented, through occlusion of the pelvis of the kidney or ureter, the pelvis and calyces of the kidney undergo dilatation to form a *retention-cyst*. The cyst may vary from the size of a pea to an extreme size, in which the kidney-substance suffers destruction from pressure and the cyst comes to occupy the entire capsule. The smaller cysts are caused by dilatation of the urinary tubules. The larger cysts, to which the name hydronephrosis is given, are due to occlusion of the pelvis of the kidney or ureter by calculi, strictures, cicatrices, and pressure from without, as from tumors of the uterus and ovaries, enlarged prostate, etc.

Other causes are cancer, cystitis, and vesical calculus. Compression of the ureter of a movable kidney may be caused by the gravid uterus. Rarely cases are caused by traumatism. Some cases are due to congenital deformity.

The **retained fluid** is usually composed of dilute urine, more or less albuminous; occasionally colloid material; sometimes blood and broken-down cells. The cysts may attain immense size. As much as thirty gallons have been removed (Glass).

The loss of one kidney may be compensated for by the opposite organ. Implication of both kidneys is always dangerous.

Treatment should address the cause. Sometimes appeal must be made to surgery : aspiration, nephrotomy, and drainage ; possibly nephrectomy.

HYPERÆMIA OF THE KIDNEY.

Hyperæmia may be acute or chronic, depending upon temporary or more or less permanent congestion of the blood-vessels of the kidney.

Acute hyperæmia of the kidney may be caused by : certain *poisons*—for instance, *cantharides*, either when ingested or sometimes when used as blisters or ointments ; the *extirpation of the opposite kidney ;* severe *traumatism ; surgical operations*, especially upon the bladder or urethra ; and *over-exertion*, forced marches, severe physical contests, and violent exercise.

The most common causes of *chronic hyperæmia* of the kidney are *chronic inflammations involving the aortic and mitral valves, dilatation of the heart, aneurism of the arch of the aorta, pulmonary emphysema, and large accumulations of fluid in the pleural cavities* that are not properly treated by removal.

In *acute hyperæmia* the urine is diminished in quantity, sometimes to constitute anuria ; the specific gravity remains about normal ; blood, albumin, and casts are present. In *chronic hyperæmia* there is some diminution in the quantity of urine, and the specific gravity may be a little higher than normal ; there is little albumin, and there are but few or no hyaline casts ; there is often dropsy, usually with some symptoms of affection of the heart.

The **treatment** will depend upon the cause. Usually of most value is rest of the kidney, which implies rest of the body, best in bed, and address to the skin and alimentary canal.

Anæmia of the kidney : In general anæmia the kidneys

may suffer a reduction in size. The quantity of urine excreted is less than normal. Chronic anæmia of the kidney is observed in arterio-sclerosis.

AMYLOID DEGENERATION.

Amyloid degeneration is most frequently recognized when the process involves the *kidneys*. Amyloid follows protracted suppuration in some part of the body, as a rule. Tuberculosis and syphilis are frequent causes.

Symptomatology and diagnosis: The patient voids an *increased quantity of clear urine*, as a rule of *low specific gravity*, containing *various amounts of albumin, with some casts and white corpuscles. Rarely there may be dropsy, especially in the lower extremities*, often associated with ascites, due to obstruction of the portal vein from affection of the liver. *Vomiting and diarrhœa* are sometimes persistent. Amyloid matter may be found in the stools, from involvement of the intestine. There may also be symptoms on the part of other organs liable to amyloid degeneration, especially the spleen and liver. Nervous symptoms are usually absent.

Evidence of amyloid degeneration may be found on the part of other organs, especially the spleen, liver, and alimentary canal. A chronic suppuration may be recognized.

Prognosis: Bad. Most may be accomplished by proper treatment of the cause of the amyloid degeneration, especially septicæmia, syphilis, tuberculosis, malaria.

Treatment: Any chronic suppuration should receive proper attention. Pus should be evacuated. Of drugs, iodine has the best reputation, probably because of the etiological relationship of syphilis. Gtt. x of the tincture, or of the ounce-to-the-ounce solution of the iodides, may be given in a wine-glassful of milk three times a day. Should digestion be impaired by iodine, the remedy may be substituted by hydrochloric acid, nux vomica, condurango, or the aromatic tincture of rhubarb.

Tuberculosis of the kidney: See Tuberculosis.

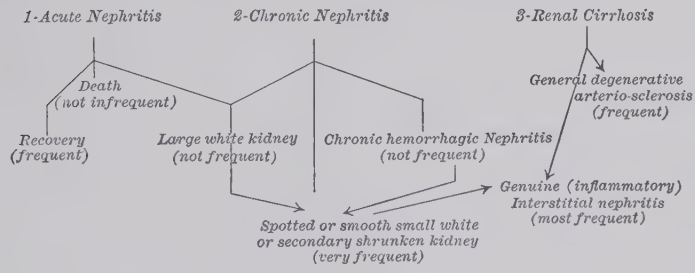
Syphilis of the kidney: See Syphilis.

NEPHRITIS; BRIGHT'S DISEASE.

Etiology: Not clear. Some investigators believe the disease to be due to micro-organisms; others attribute it to toxins or chemical poisons. Probably both views are correct. Some cases are caused by ptomaines, toxalbumins, acetone (von Jaksch); and cases may be caused by uric acid, creatin, xanthin, and also by cantharides and other poisons. *Interstitial nephritis* has been produced experimentally, in the dog, by the subcutaneous injection of oxalic acid and oxamide (Ebstein and Nicolaier).

Most cases of nephritis are attributed to the infections, including "colds." Pregnancy often plays a prominent *rôle*.

Classification of nephritis: After Pel, of Amsterdam, as given by Whittaker:



Three varieties of Bright's disease are generally accepted: acute nephritis, chronic nephritis, and renal cirrhosis. Besides these there are transition-forms, as indicated in the above table.

Acute Parenchymatous Nephritis.

Etiology: Acute parenchymatous nephritis is caused by the excretion of some poison through the kidneys. Typical acute inflammation of the kidney may be produced by cantharides. Acute parenchymatous nephritis is most frequently caused by toxins, toxalbumins, in the course of or following the infec-

tious diseases, especially scarlet fever and diphtheria; less frequently measles, rötheln, smallpox, pneumonia; rarely typhoid fever and the other infections. Some cases are ascribed to "cold" and pregnancy. "Colds" are usually infectious.

Symptomatology: The onset may be sudden or insidious. Usually *micturition is increased in frequency*, but the *quantity of urine voided in twenty-four hours is less than normal.* There may even be anuria. With the reduction in quantity there is an increased specific gravity, 1025–1030. There may be hæmaturia with consequent change in color of the urine. More characteristic is the presence of *albumin*, usually with *epithelial and blood-casts.* As a rule there is *dropsy*, observed first as a puffiness *about the eyelids*, sometimes extending to become general over the body, possibly to take life through œdema of the lungs or glottis. Prominent nervous symptoms are *headache and neuralgia, vertigo, nausea and vomiting.* Some cases are announced by *sudden blindness* or *early convulsions.* There may be sopor, stupor, and coma.

Chronic Parenchymatous Nephritis.

Etiology: Chronic parenchymatous nephritis is caused, for the most part, by the long-continued elimination of a poison, usually a toxin. Some cases result from acute parenchymatous nephritis, especially when due to septicæmia, syphilis, or tuberculosis; sometimes when due to scarlatina, pregnancy, or "cold."

Symptomatology: The onset is usually insidious, with *loss of ambition, fatigue on slight exertion*, sometimes with *nervous symptoms*, especially *hebetude, headache, and neuralgia.* There are anorexia, *loss of weight, pallor, drowsiness or insomnia*, palpitation, shortness of breath; and *dropsy*, appearing first as *œdema of the face*, especially the eyelids; and about the ankles, becoming later extensive and marked. There may be *retinitis albuminurica.*

The *urine is reduced in quantity*, from two pints to a half pint or less in twenty-four hours, and is *high in specific gravity*, 1025–1040, and cloudy. *Albumin* is present in large quantity,

and *casts* may be found in large numbers and great variety. *Broad, waxy,* and granular casts are characteristic.

Vomiting and diarrhœa may become troublesome and persistent. The poison of the disease seems sometimes to become localized in an *inflammation of some serous membrane,* as a pleuritis, peritonitis, or pericarditis. *Nervous symptoms* may be present in all grades of severity.

Renal Cirrhosis.

Renal cirrhosis is the most frequent form of Bright's disease, constituting more than one-half of all cases. The onset is insidious, so that the condition may be unrecognized for a number of years. Renal cirrhosis is characterized by the secretion of a large amount of urine, of light specific gravity; the presence of nervous symptoms, and the absence of dropsy.

Symptomatology: *The onset of renal cirrhosis is insidious,* with *depression of spirits* and *impairment of health.* The color is bad. *Nervous symptoms predominate.* There are often headache, neuralgia, vertigo, dyspnœa (renal asthma), *palpitation* of the heart, and sometimes *blindness* or other disturbance of vision. The first suspicion of the disease may be aroused by the occurrence of apoplexy or hemorrhage, especially from the nose, stomach, or bowels. The patient voids a *large quantity of clear urine, with light specific gravity,* 1010 or less. The urine contains little sediment. Upon examining a specimen from the total quantity passed in twenty-four hours a *trace of albumin* will be found (see Spiegler's test). *Casts* are few and difficult to find. *Usually there is no dropsy.* Enlargement of the heart is caused by *hypertrophy of the left ventricle,* which becomes necessary to overcome the obstruction offered to the circulation through the kidneys. The strong heart continues for a long time to force a large quantity of fluid through the kidneys, and as long as this continues dropsy is absent. The urine, though passed in large quantities, contains little solid matter, so that symptoms of uræmia are not infrequent.

Diagnosis of Bright's Disease.

Acute parenchymatous nephritis may come on suddenly or insidiously. There is a reduction in the quantity of urine, which is high in specific gravity and contains albumin and casts, especially epithelial and blood-casts. Œdema appears first about the eyelids. There may be headache, neuralgia, vertigo, convulsions, nausea and vomiting, sometimes sudden blindness.

In the presence of the *large white kidney* there are marked pallor and obstinate dropsy, in the absence of hypertrophy of the heart. The urine is small in quantity, of high specific gravity, and contains casts, especially fatty and granular casts. Nervous symptoms are usually not pronounced.

Chronic parenchymatous nephritis is usually insidious in onset, with general degradation of the health, spirits, and strength. Some cases show nervous symptoms. There is œdema, first of the face and ankles. A retinitis albuminurica may be observed. The urine is reduced in quantity, of high specific gravity, and contains albumin and casts, especially broad and waxy casts. There may be inflammation of the serosæ. Often there are vomiting and diarrhœa. Nervous symptoms vary in severity.

In **hemorrhagic nephritis** there is persistent bleeding. The course is longer than in acute nephritis, and the nervous symptoms are not so marked.

Cases of the *small white kidney* show enlargement of the heart, and reduced quantity of urine containing casts of all kinds. Transition from the large white kidney, or chronic hemorrhagic nephritis, to the small white kidney is marked by an increase in the quantity of urine and diminution in the number of casts, with hypertrophy of the heart.

Renal cirrhosis, the most frequent form of Bright's disease, begins insidiously. Nervous symptoms are marked. Œdema is slight or absent. A large quantity of urine is voided, of light specific gravity, containing a trace of albumin and possibly a few hyaline casts. There are hypertrophy of the heart and the symptoms of arterio-sclerosis. Blindness may be caused by retinitis albuminurica. The patients are usually in middle or advanced life.

Prognosis of Bright's Disease.

Acute nephritis may result in recovery. Anuria, hæmaturia, and severe nervous symptoms, especially convulsions and coma, are ominous signs. Much depends upon the time when proper treatment is instituted.

Cases of **chronic nephritis** or **renal cirrhosis** probably never recover, but the patients may live for a long time under proper treatment and hygiene.

Treatment of Bright's Disease.

The best single article of **diet** is *milk*, which may be used exclusively in severe cases. Later, *vegetables* and *fruit* may be added. The patient should drink an *abundance of pure water;* but an undue amount of fluid must not be given in the presence of a weak heart. *Meat* may be given, but not to excess: The patient may receive stewed sweetbreads, stewed chicken, calf-brains, pig's feet, or broiled fish once a day.

Bathing: A *hot bath*, 100°–110° F., may be given at bedtime, and in severe cases oftener. Duration of bath, five to twenty minutes. A hot drink should be given before and after the bath. While in the bath the patient's head should be enveloped in a cloth wrung out of cold water. Sometimes it is necessary to substitute the hot bath by the hot pack. Palpitation and a fluttering heart are *contraindications* to the hot bath. In such cases pilocarpine may be used, gr. $\frac{1}{12}$–$\frac{1}{4}$, subcutaneously.

Drugs: *Pilocarpine* has been recommended externally, in the form of an ointment, 0.05–0.1 to 10.0 of vaseline (Mollière). The use of pilocarpine is contraindicated by uræmia.

Cardiac diuretics, usually contraindicated in acute parenchymatous nephritis, are often of service in *chronic nephritis* and *renal cirrhosis*. The best diuretic is digitalis, which at times may be substituted by strophanthus, sodium-benzoate of caffeine, diuretin, or nitroglycerin, which is the best agent with which to secure immediate results in the presence of cirrhosis.

Dropsy that causes dyspnœa or other great discomfort, or that threatens rupture of the skin, may be relieved by the free use of calomel or a dose or two of elaterin; or by the introduction of silver canulæ into the feet or legs, or by minute punctures of the skin.

Often a **change of residence** to a warm, dry climate is advisable. The patient should wear warm clothing and remain indoors during inclement weather.

Further treatment is addressed to the *relief of symptoms.*

CYSTITIS.

Etiology: Cystitis is caused most frequently by extension of disease from the urethra, especially gonorrhœa. Next most frequently, the cause of cystitis comes from above, especially from pyelitis and calculus.

Tuberculosis and cancer may be conveyed to the bladder through the blood or lymph-vessels, or by extension from contiguous structures, especially from the uterus and rectum.

Infection of the bladder may occur as a local expression of septicæmia. Paralysis of the bladder, by permitting retention, may favor infection of the bladder. Sometimes trauma, especially the introduction of foreign material into the bladder, plays an important part in the etiology of cystitis. In operations upon the bladder, the use of the catheter and the introduction of the cystoscope or instruments for crushing and removing stones, strict asepsis should be observed. Stricture of the urethra, or any cause of retention of urine, is a prominent cause of cystitis.

Cystitis—symptomatology: There are *pain* in the region of the bladder, *tenderness*, vesical and rectal *tenesmus*, *frequent and painful micturition*. The *urine* contains *pus*, *mucus*, frequently blood, and usually large numbers of the characteristic flat *epithelial cells* found normally in the bladder. The pain is often reflected to the penis, testicles, and rectum. In tuberculosis of the bladder there is often polyuria. When cystitis is due to the presence of a calculus in the bladder, there is often hemorrhage, and pain is increased by movement of the body.

Prophylaxis: Only clean instruments should be introduced into the bladder. This applies equally to the use of the catheter, cystoscope, and instruments for crushing stones. Urethritis, especially gonorrhœa, should receive early and continuous treatment until completely cured. Strictures should be dilated and stones removed.

Cystitis—treatment: Rest in bed is important. The diet should be light. The bowels must be kept open. The bladder may be flushed by the free use of hot drinks, milk, mucilaginous drinks. Harrison recommends a combination of the ulmus fulva, or slippery elm, and succus hyoscyami in decoction. Hyoscyamin may be given, gtt. iij–v of the grain-to-the-ounce solution, every three or four hours. Atropine is administered in the same dosage.

Great relief is often afforded by the warm bath, and the rectal injection of hot water. More severe pain calls for opium in suppositories or by rectal injection. Strangury may be relieved by the subcutaneous use of morphine, preferably suprapubic, in the region of the bladder.

Cystitis due to gonorrhœa is benefited by the internal use of copaiba, the oil of yellow sandalwood, salol, the salicylate of sodium, and methylene-blue.

Chronic cases of cystitis are best treated by irrigation of the bladder. For this purpose use may be made of solutions of nitrate of silver; protargol; creolin; boric acid; bichloride of mercury (1: 20,000); permanganate of potassium; tannin; sulphate of zinc; alum; alumnol, or the neutral sulphate of quinine (gr. j to ℥j).

ENURESIS (Incontinence of Urine).

The **involuntary discharge of urine** occurs normally in infancy. Sometimes delayed development of the sphincter, especially of its nerve-supply, causes persistence of enuresis. Thus the condition is seen frequently from three to ten years of age, especially during sleep.

As a *neurosis*, enuresis occurs in the presence of a normal bladder and normal urine. At times the condition appears at puberty, often with other neuroses, sometimes onanism.

Inability to retain the urine—incontinence of urine—occurs most frequently in the young and old; in women more often than in men. In women the urethra may suffer dilatation and the action of the sphincter be imperfect, especially after parturition, so that urine may be discharged with no, or but slight strain, such as coughing, sneezing, laughing.

Enuresis may be caused by masturbation. Men, especially in old age, suffer incontinence of urine through affection of the prostate.

Incontinence of urine is caused by *overdistention* of the bladder, or by defective closure of the sphincter. Thus, the condition may be caused by either paresis of the detrusor or of the sphincter. Sometimes incontinence of urine results from organic disease.

Treatment: An overdistended bladder should be relieved with a clean catheter. Urethral polypi and calculi should be removed. Often much may be accomplished with electricity, particularly when the current is brought into contact with the sphincter by introducing the electrode into the rectum. The best drug is atropine, gtt. iij of the grain-to-the-ounce solution at bedtime, increased gradually up to tolerance.

In the way of general treatment, cold baths, outdoor exercise, and the use of iron, quinine, and cod-liver oil are of value.

SPERMATORRHŒA.

The **involuntary discharge of semen** may occur physiologically once or twice a month. The condition is pathological, only when the discharge is followed by exhaustion. But not every discharge of fluid, even when followed by exhaustion, is a spermatorrhœa. The fluid may not be semen. Thus the condition may be a **prostatorrhœa**.

Etiology: The most frequent causes of spermatorrhœa are masturbation, sexual excess; urethritis, especially gonorrhœa; an elongated prepuce, phimosis; ascarides, constipation, hemorrhoids, eczematous eruptions, and abnormal conditions of the rectum and anus.

Symptomatology: Sometimes the patient is not aware of the loss of semen, which is discharged with the urine, possibly to

be recognized as a cloudiness of the urine and the discovery of spermatozoa upon microscopic examination. But spermatozoids are not always present in semen; they may be absent in *impotence.*

Following a *pathological discharge of semen* there are exhaustion, a *feeling of weakness, languor, and depression,* and various *nervous disturbances, paræsthesiæ, flashes of heat, headache, and vertigo.* Later there are *palpitation, dyspnœa, and dyspepsia.* Depending usually upon sexual abuse, there is as a rule more or less hypochondriasis and melancholia.

Diagnosis: In the presence of an exhausting discharge, the clinician should determine whether or not the discharge is semen. A simple prostatorrhœa will not contain spermatozoids. Sometimes the discharge of semen takes place with urination, when the spermatozoids may be found in the urine.

Treatment: Sexual abuse should be stopped. The sexual function had better be given a rest and the mind occupied with healthy thought and the body with healthful exercise. The cold bath or cold douche invigorates the body. Electricity is sometimes of value: short sessions of feeble galvanic or faradic currents.

Posterior urethritis should be properly treated. Matrimony may be advisable, when other treatment, as a rule, becomes unnecessary. The discharge of semen, which usually occurs at night, may be prevented by potassium bromide, gr. xx–xl, largely diluted, at bedtime.

IMPOTENCE.

Incapacity for natural coitus may result from lack of sexual desire, absent or imperfect erection, premature discharge and alterations, especially reduction or absence, of the seminal fluid, and the absence of living spermatozoids.

Impotence is caused most frequently by excess in venery, onanism, and gonorrhœa. The condition occurs early in diabetes, diphtheria, and locomotor ataxia. Aversion, dislike, and the fear of loss of the sexual power are prominent physical causes. Toxic causes are alcohol, the bromides, iodides, opium, camphor, salicylic acid, and lupulin. The condition

may be caused by physical malformations and defects, atrophy, and tumors. Undescended testicles cause impotence only when atrophied.

The **prognosis** depends upon the cause, and is usually most favorable in the toxic and psychical cases.

Treatment: The cause must be removed. Rest and abstention are important. The best remedies, as a rule, are strychnine and electricity.

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